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INFECTION OF THE RENAL PARENCHYMA FROM THE PELVIS OF THE KIDNEY

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Although there is still a great difference of opinion regarding the usual mode of infection in bacillary disease of the urinary tract, it is generally admitted that infection of the pelvis and of the renal parenchyma does occur by the ascending route. In many instances the process is aided by obstruction in some portion of the urinary tract. A vast amount of experimental work has been done in tracing the infection from the bladder to the renal pelvis, and it has been shown that it can travel up the periureteral lymphatic structures and that infection of the pelvis can come from the bladder by way of the lumen of the ureter without any evidence of involvement of the ureteral wall.

I shall not consider the mode of infection of the renal pelvis. It is my object to show that stasis in the urinary passages is the determining factor in the breaking through of an infection from the pelvis of the kidney to the parenchyma of the kidney. Pathologic studies on human subjects help little in solving the problem because at necropsy the renal parenchyma is usually seen to be so intensely and widely involved that the route of infection is completely obliterated. Only occasionally are early lesions seen in human kidneys which are comparable to those produced experimentally in animals.

There are at least four ways by which infection may enter the renal parenchyma: (1) by ascent of the collecting tubules, producing local lesions in the papilla, medulla or cortex; (2) by extension along the perivascular lymph spaces of the large vessels entering the substance of the kidney; (3) by the vascular route, either arterial or venous, by direct infection from the adjacent infected pelvis, and (4) by direct extension through the wall of the pelvis into the renal parenchyma at any point where the parenchyma lies in direct contact with the pelvis, a point frequently involved being the place where the pelvic lining is reflected from the renal parenchyma.

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In addition to the lymphatic and hematogenous routes, Hinman [1] mentioned the pyelovenous, tubular, tubulovenous and tubulolymphatic routes, or the interstitial back-flow. He stated that when back pressure is present and infection of the blood stream has occurred, lesions may be produced when the organisms are excreted. In view of the presence of organisms in the renal tissue, it seems hardly necessary to assume a complete circuit of the infectious agent through the circulatory system and its return to the substance of the kidney. Hinman,[1] as well as many others, stated that infection of the renal parenchyma from the pelvis is not likely to occur without some form of obstruction. The significance of this is evident when one realizes how difficult it is to produce infection of the renal parenchyma without back pressure.

On the basis of the work done by Schmidt and Aschoff,[2] MacCallum [3] considered the first mode of infection the most likely one. Their experiments were with rabbits which had complete obstruction of the ureters. They laid great emphasis on necrosis of the papillae, which, if one judges by their work as well as by my experimental work with sterile hydronephrosis, is definitely anemic necrosis resulting from the cutting off of the blood supply. Necrosis of the papillary tubules may be observed even after twenty-four hours. As a result of experimental work as well as of study of postmortem material, Kennedy [4] rejected the theory of ascent of the infection by way of the tubules and favored the perivascular route.

Müller,[5] as a result of examination of human pathologic material only, came to the conclusion that infections enter the parenchyma of the kidney by way of the perivascular lymphatics, through extension between the tubules and finally through rupture of inflammatory products into the lumen of the tubules. Histologic studies in cases of the most acute infection, in one of which, for example, death occurred on the fifth day, have left me doubtful as to the course of the infection in children.

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1. Hinman, Frank: The Principles and Practice of Urology, Philadelphia, W. B. Saunders Company, 1935.

2. Schmidt, M. B., and Aschoff, Ludwig: Die Pyelonephritis in anatomischer und bakteriologischer Beziehung und die ursächliche Bedeutung des Bacterium coli commune für die Erkrankung der Harnweg, Jena, Gustav Fischer, 1893.

3. MacCallum, W. G.: A Text-Book of Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1932.

4. Kennedy, R. L. J.: The Pathologic Changes in Pyelitis of Children Interpreted on the Basis of Experimental Lesions, J. Urol. **27**:371-398 (April) 1932.

5. Müller, Achilles: Untersuchungen über die Ausbreitung des eutzündlichen Processes im Nierenparenchym bei der aufsteigenden Pyelonephritis, Arch. f. klin. Chir. **97**:44-73, 1912.

The present study can be divided into two parts, the first dealing with the spread of infection in the presence of obstruction in the ureter and the second dealing with the changes produced in the kidneys in the absence of ureteral obstruction.

The experiments in the first part pertained to the parenchymal lesions produced when Escherichia coli or Staphylococcus aureus was injected into the ureter and the ureter was then tied above the point of injection. In view of the fact that the experiments with these two organisms corresponded closely, only those with Esch. coli will be discussed.

The intervals between the injection and the termination of the experiment were eighteen, twenty-four, thirty, thirty-six, forty-eight and ninety-six hours. As will be seen in table 1, five experiments were done with an eighteen hour interval and six with a twenty-four hour interval. These two sets of experiments, in which the interval was short, were the most conclusive as well as the most

TABLE 1.—*Parenchymal Lesions Following Injections of Bacillus Coli Into Obstructed Ureter*

Time, Hr.	Number of Experiments	Infection		Venous Thrombosis	Abscess	
		Peritubular	Perivascular		Cortex	Medulla
18	5	3	3	2	0	0
24	6	4	5	3	2	0
30	5	3	3	5	1	0
36	4	2	3	3	1	1
48	4	4	4	4	4	4
96	1	1	1	1	1	1

interesting of all because they indicated the route of the infection in its earliest stages, before the various parts of the kidney had become involved. The lesions were essentially peritubular, perivascular and vascular and, in contradistinction to the findings of Schmidt and Aschoff, were entirely absent from the medulla. The cortex in two instances showed abscesses after the twenty-four hour interval. After eighteen hours the inflammatory process could be traced to the cortex along the perivascular lymphatics (fig. 1). In figure 2, which also shows the results of an eighteen hour experiment, direct extension through the pelvic wall is seen at the point of reflection of the pelvic lining from the renal parenchyma. In this area thrombosis of a large vein is observed. At this stage the papilla and the medulla of the kidney were entirely free from inflammatory reaction.

At the end of thirty hours one observed that in addition to the foregoing changes the perivascular, peritubular and thrombotic lesions were the outstanding features, but there were no changes in the medulla. The first abscess in the medulla was observed after thirty-six hours. After forty-eight hours, in the four experiments done, there were peritubular, perivascular and vascular lesions and abscesses in the medulla and the cortex. In other words, there was complete involvement of the parenchyma of the kidney within forty-eight hours when the ureter had been completely obstructed. Thus, only in experiments in which the

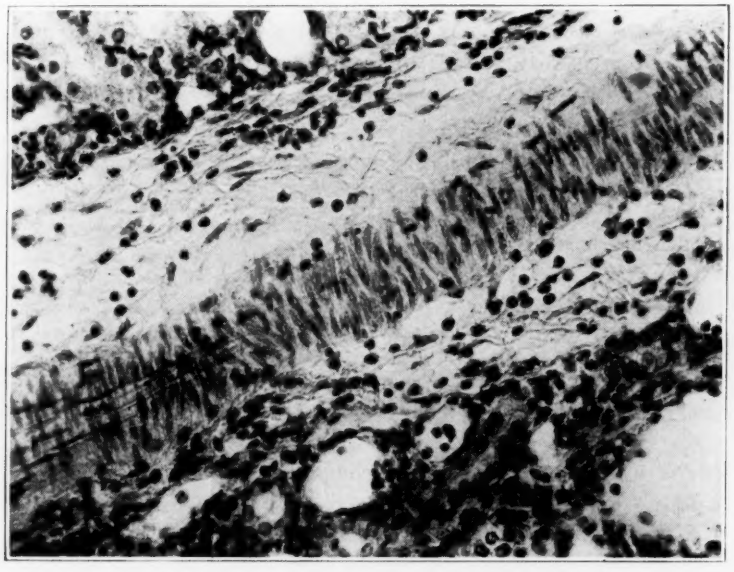

Fig. 1.—Acute polymorphonuclear perivascular inflammation in the cortex.

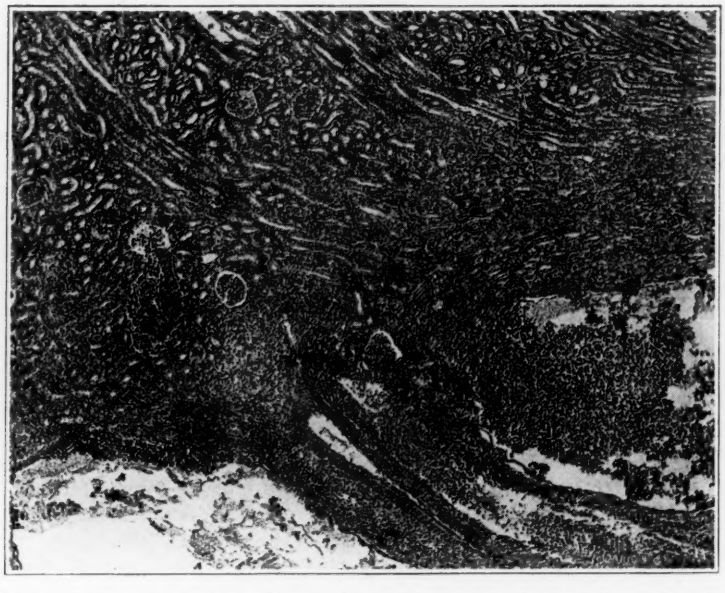

Fig. 2.—Rupture of the pelvic lining, direct extension of the inflammation into the renal parenchyma and thrombosis of the vein.

interval was less than forty-eight hours was it possible to determine by what route the various portions of the parenchyma had become infected. There was widespread distribution of lesions after ninety-six hours (fig. 3).

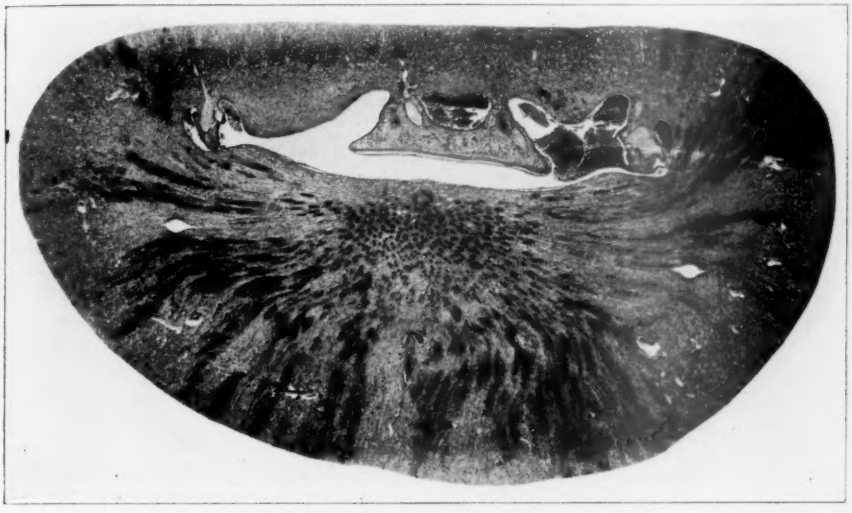

Fig. 3.—Cross-section of a kidney ninety-six hours after an injection into the obstructed ureter, showing diffuse lesions of the cortex, medulla and papilla.

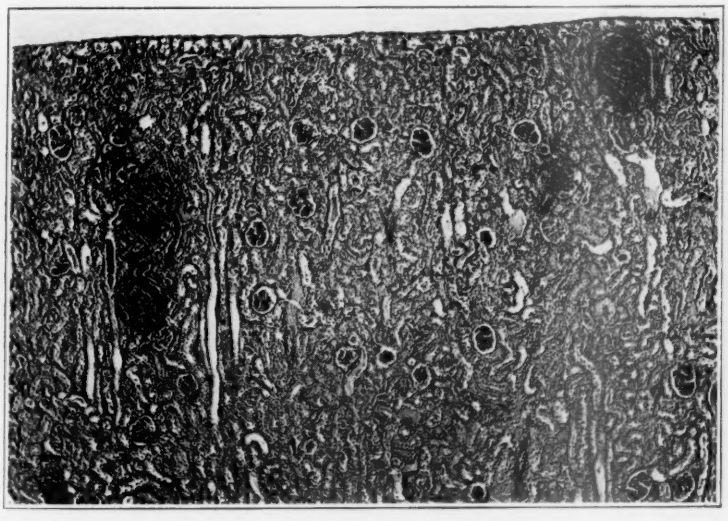

Fig. 4.—Cortical abscesses eighteen hours after an injection into an obstructed ureter.

In table 2 the same development of lesions is shown for staphylococcic infection. In this series of experiments cortical abscesses were first seen after eighteen hours (fig. 4).

It is thus evident that infection can enter the parenchyma by way of the perivascular lymphatics, by direct extension through the pelvic wall and by direct involvement of the vessels in the peripelvic tissue. The frequency and the extent of thrombosis of the renal vessels make infarction an added factor in the destruction of renal tissue. Focal lesions appeared first in the cortex as early as after eighteen hours, and these lesions were followed by lesions in the medulla and the papillae. Whether infection ever spread by ascent of the collecting tubules must be left undecided, as in this series of experiments no evidence of such ascent was found. The severe inflammatory reaction in the peripelvic tissues and the constant vascular lesions make the entrance of organisms into the blood stream easy, and it is likely that bacteremia is the rule rather than the exception in cases of this type of infection.

In a study of infection of the renal parenchyma from the pelvis when there was no ureteral obstruction, a different picture was found. The lesions were few and were often so extensive that the route of

TABLE 2.—*Parenchymal Lesions Following Injections of Staph. Aureus Into Obstructed Ureters*

Time, Hr.	Number of Experiments	Infection		Venous Thrombosis	Abscess	
		Peritubular	Perivascular		Cortex	Medulla
18	4	1	1	2	0	0
24	8	3	3	5	2	0
30	3	1	2	3	1	0
36	4	3	4	4	1	1
48	6	3	5	3	2	0

infection was practically obliterated. The usual picture was that of acute pyelitis, more marked on the parietal surface, with the renal parenchyma offering a definite barrier to infection. In only one of the fifteen experiments in which my co-workers and I attempted to produce pyelitis by injection of colon bacilli into the bladder were we able to produce a renal lesion, which consisted of a single group of abscesses in each kidney (fig. 5). At a later date, by injection into the bladder of a number of virulent strains of Esch. coli, together with foreign bodies, we were able to produce pyelitis in four of eight rabbits (table 3); but in only two of these were we able to produce any changes in the parenchyma of the kidney. As in the experiments with obstruction, the lesions were in the nature of perivascular infiltrations along the large vessels (fig. 6), venous thrombi and peritubular infiltrations extending out from the thrombotic areas (fig. 7). In spite of intensely inflammatory lesions in the peripelvic tissues, amounting in some areas to actual abscesses and multiple mural thrombosis of the veins (fig. 8), no definite abscesses were seen in the renal parenchyma. The papillae and the medulla were free from inflammation. Along the large vessels

were inflammatory lesions, and the bacteria extended into the cortex of the kidney. In a number of areas of multiple venous thrombosis the intertubular inflammatory reactions were intense.

The series of experiments in which spontaneous pyelitis developed in rabbits furnished only one example of extensive involvement of

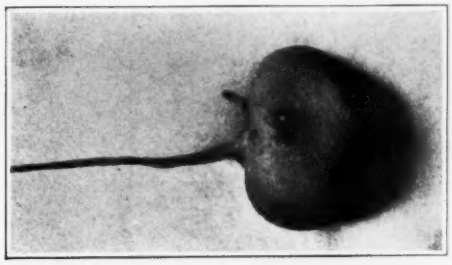

Fig. 5.—A single group of cortical abscesses produced by ascending infection following an intracystic injection.

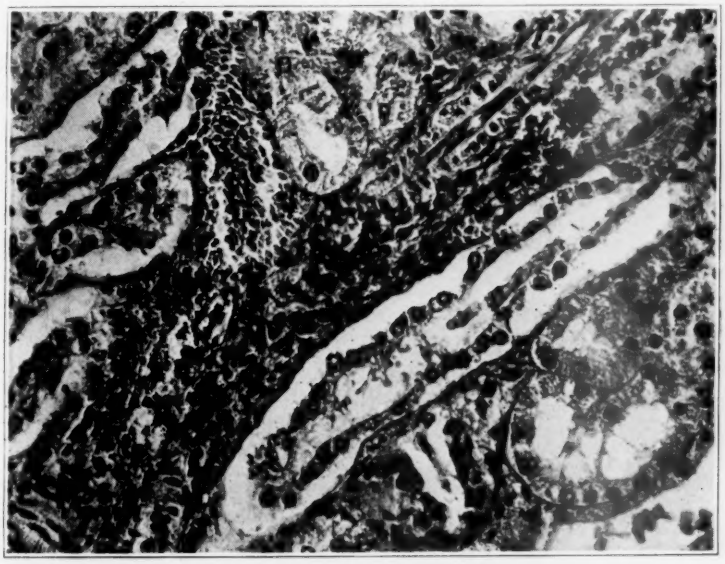

Fig. 6.—Acute perivascular infiltration of the cortex after an intracystic injection.

the renal parenchyma. The lesion in these experiments, as in the experiment in which an abscess of the cortex was produced by cystic injection, was a single large group of abscesses (fig. 9). The nature of the path of the infection from pelvis to parenchyma could not be made out, but the similarity of the lesions is interesting, especially as there was a similar one in the animals that had received ureteral injections.

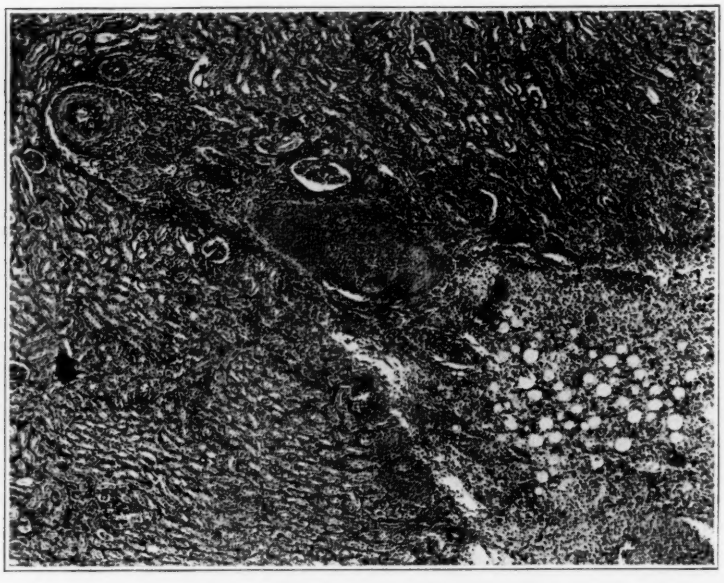

Fig. 7.—Multiple thrombi and intertubular infiltration into the kidney after an intracystic injection.

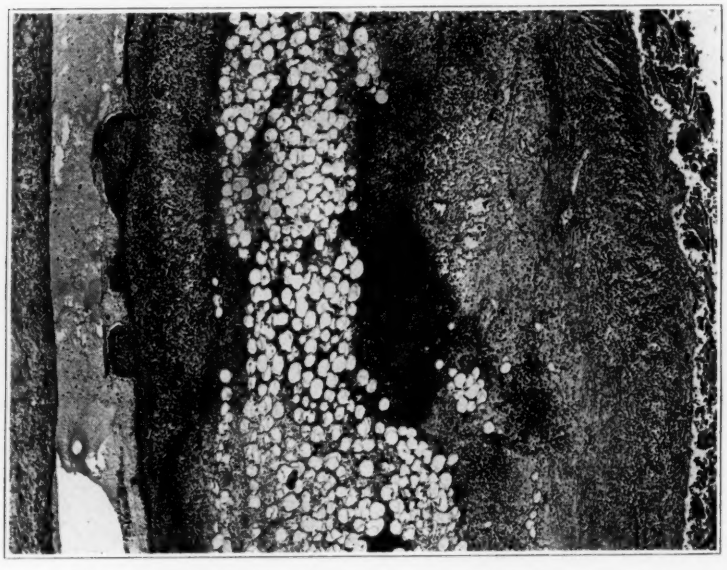

Fig. 8.—Multiple mural thrombi in the renal vein of the peripelvic tissues after an intracystic injection.

The uncertainty of producing pyelitis by intracystic injection interrupted our experiments for a number of years. Recently, however, through the cooperation of Dr. Mann and his staff, a series of rabbits and dogs in which the bladder had been transplanted into the anterior abdominal wall were made available. These animals furnished ideal material for the continuation of our experiments. Ureteral urine could

TABLE 3.—*Lesions of the Urinary Tract After Intracystic Injection of Cultures of Different Strains of Colon Bacilli*

					Peripelvic Tissue		Renal Parenchyma				
Rabbit	Kidney	Age of Lesion, Days	Periureteral Inflammation	Pyelitis	Inflammation	Thrombosis	Perirenal Inflammation	Focal Lesions of Papilla	Perivascular Infiltration	Venous Thrombi	Cortical Infiltration
1	Right	3	0	++	0	0	0	+*	0	0	0
	Left		+	++++	+++	+++	+	+*	+	++	0
2	Right	3	0	+++	++++	+++	++	+**	0	0	0
	Left		+	++++	++++	++	+++	+**	0	0	0
3	Right	4	+++	+	++	0	0	0	0	0	0
	Left		0	0	0	0	0	0	0	0	0
4	Right	5	++	+++	+++	++	++	0	+++	+++	0
	Left		0	..	0	0	0	0	0	0	0

* Bacterial thrombi in papillary vessels, cortex and glomeruli.
** Subepithelial infiltration.

TABLE 4.—*Infection of the Kidney in Dogs with Exstrophy Following Ureteral Injection*

Experiments		Infection of Kidney			
			Parenchymal Lesion		
Duration, Days	Number	Pyelitis	Peripelvic	Papilla	Cortex
2	2	2	2	1*	1*
3	1	1	1	0	0
4	1	1	1	0	0
5	6	6	6	1†	1†
6	3	3	3	0	0
21	1	1	1	0	0
39-54	4	4	2	0	0

* Extremely sick dog, with mild pyelitis.
† Pus cells in the urine before injection; one large group of lesions.

be obtained directly from each kidney, and a bacterial suspension could be injected into the pelvis of the kidneys.

In the first experiments, only one ureter received an injection, and the other kidney was kept as a control. After we saw that a number of control kidneys were normal, we used each animal for two experiments, injecting the material into one kidney on a certain day and into the other a few days later. In this manner eighteen experiments were carried out. Table 4 shows the results of these experiments.

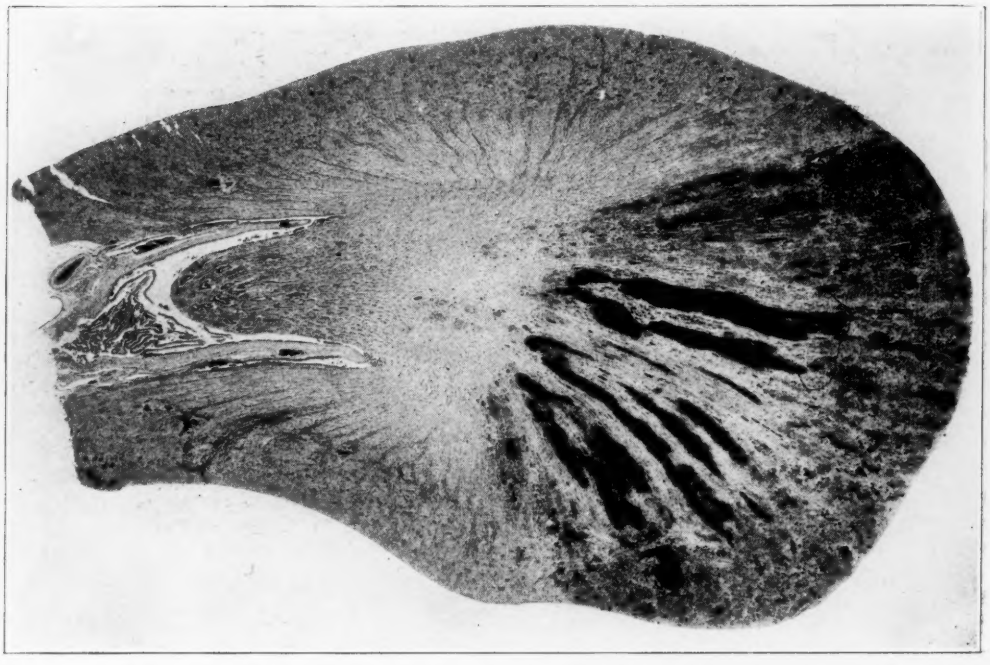

Fig. 9.—A single large group of abscesses associated with spontaneous pyelitis in a rabbit.

Fig. 10.—A single large multilocular abscess following an intra-ureteral injection.

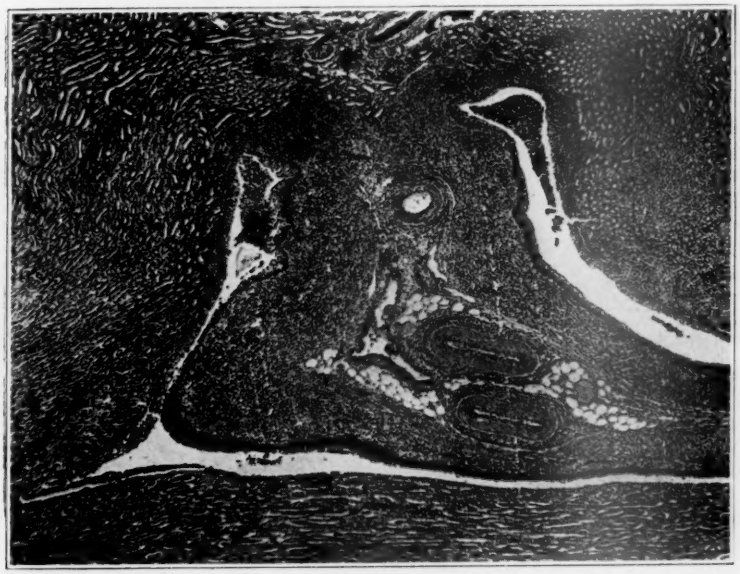

Fig. 11.—Pyelitis, especially inflammation of the visceral layer, following an intra-ureteral injection.

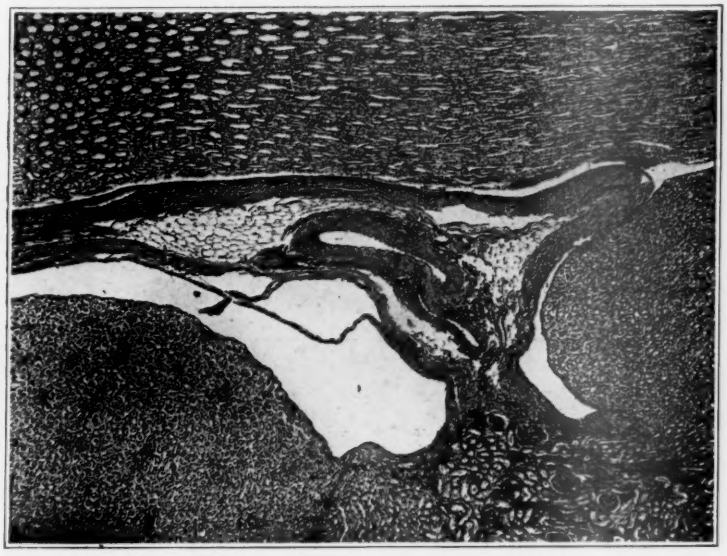

Fig. 12.—Section of a control kidney; the other kidney is shown in figure 11.

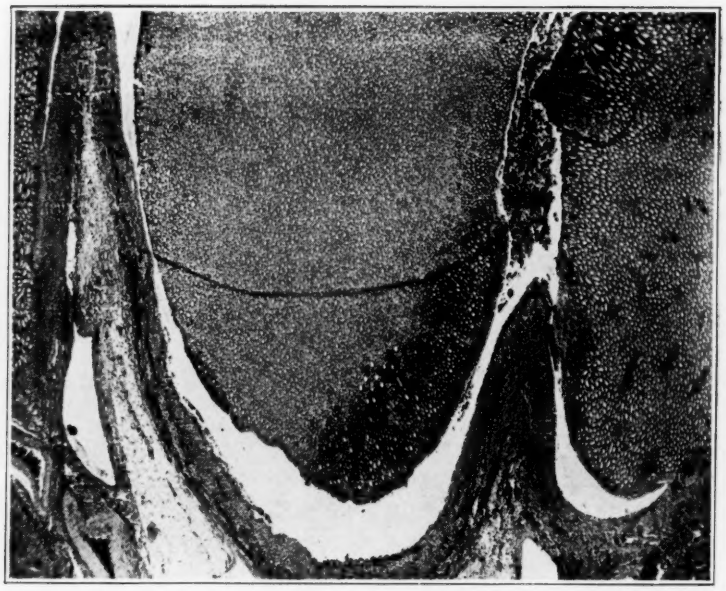

Fig. 13.—Pyelitis, extensive peritubular infiltration and hemorrhage of a papilla.

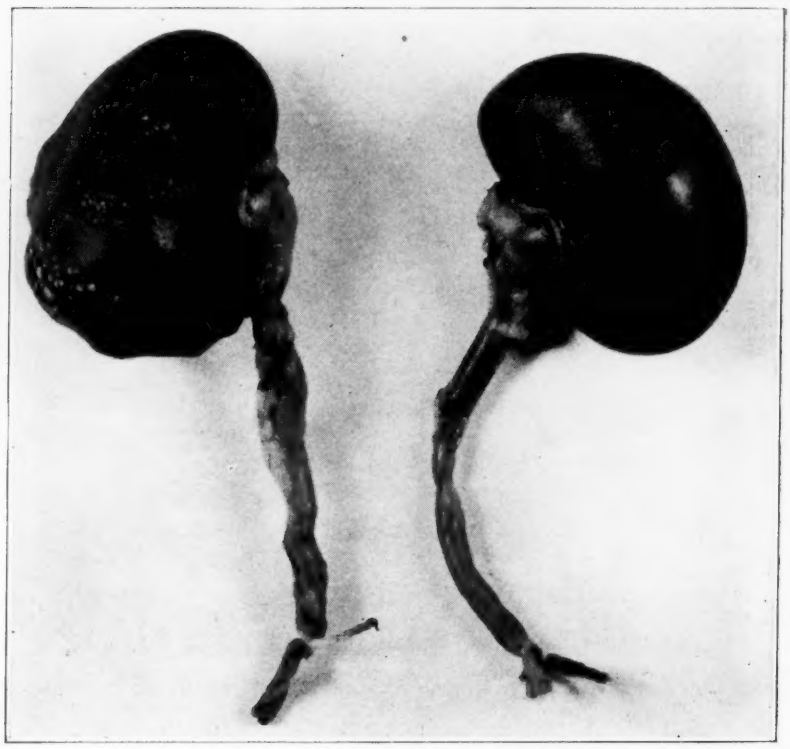

Fig. 14.—Multiple abscesses complicating hydronephrosis in the cortex of the kidney of a rabbit after an intra-ureteral injection.

The striking feature was the absence of parenchymal lesions in the kidney. In only two animals were there definite lesions at a distance from the pelvis. In one animal, a dog that had to be killed because it was extremely ill, we observed multiple abscesses in the medulla and the papilla of the kidney five days after the first injection and two days after the second injection, but these apparently were not related to the mild inflammation of the pelvis of the kidney. In the other animal, one kidney exhibited lesions which were limited to a single large group of abscesses (fig. 10), which closely resembled the single cortical abscess produced by intracystic injection as well as the single group abscess observed in the rabbit with spontaneous pyelitis.

In the other sixteen experiments the lesions, although extremely severe and acute, were limited to the immediate vicinity of the pelvis. The epithelium showed many cysts filled with pus cells, but no vascular thrombi were seen. The parietal side of the pelvis was edematous and densely infiltrated with many polymorphonuclear leukocytes and young connective tissue cells (fig. 11). The absence of inflammatory reaction in the opposite kidney, used as a control, is seen in figure 12. The visceral side of the pelvis showed much less infiltration, and often this process was entirely absent. In only one instance were there considerable intertubular

TABLE 5.—*Infection of the Kidney in Rabbits with Exstrophy Following Ureteral Injection*

Experiments		Infection of Kidney			
			Parenchymal Lesions		
Duration, Days	Number	Pyelitis	Peripelvic	Papilla	Cortex
3	1	1	1	1*	1*
4	1	1	1	0	0
5	5	5	5	0	0

* Hydronephrosis.

infiltration and hemorrhage (fig. 13). Of the seven rabbits, only one showed lesions in the parenchyma (table 5), and in this animal, owing to a faulty operative procedure, definite hydronephrosis had been produced in both kidneys. The kidney on the side of the injection showed extensive studding with cortical abscesses, and the control kidney showed similar hydronephrosis without the abscesses (fig. 14). The other animals showed only inflammatory reaction in the lining of the pelvis.

This group of experiments shows characteristically that, even with the intense pyelitis produced by direct injection of bacteria into the pelvis of the kidney, a barrier exists to the spread of infection into the renal substance. These experiments bring out most clearly that in the unobstructed urinary tract such an entity as pyelitis can exist, that the kidney has a protective mechanism by which the spread of infection is kept out of the kidney proper and that this barrier seemingly remains intact as long as there is no obstruction to the outflow of urine. It is broken down in less than twenty-four hours when there is obstruction. A series of experiments with more highly virulent organisms might show a different result, although one of the organisms used was isolated from the urine of a dog suffering from severe pyelitis.

These experiments emphasize in a drastic way the importance of maintaining adequate drainage in all cases of infection of the urinary tract, and they show what has been emphasized so often, that in the acute stages of urinary infection the all-important object of treatment is adequate production of urine to wash out the thick, purulent exudate and so prevent stasis.

SUMMARY AND CONCLUSIONS

With stasis, the parenchyma of the kidney rapidly becomes infected from the pelvis by way of the perivascular lymphatic structures and by direct passage of the organisms through the pelvic lining over the parenchyma. Thrombosis forms an important feature of the renal changes. Without stasis, infection of the pelvis tends to remain local, and only exceptionally do changes occur in the parenchyma.

The striking difference between the two series of experiments shows the importance of avoiding stasis at all costs in the treatment of ascending infection of the urinary tract.

FATE OF CHILDREN INFECTED WITH TUBERCULOSIS DURING THE FIRST FIVE YEARS OF LIFE

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There is increasingly less pessimism concerning the fate of children infected with tuberculosis during infancy. The reason for this change lies partly in the progressively improving ability of the medical profession to diagnose mild and subclinical tuberculosis in infants and young children. Before the discovery of the tuberculin test and the roentgen rays, only those infants who presented clinical signs were regarded as tuberculous. The prognosis of such infants was and still is grave. However, apparently well children whose only sign of tuberculous infection is a positive reaction to the tuberculin test or an abnormal appearance of the lungs in a roentgenogram of the chest are now known. They really belong in a different category than do the clinically ill children and have a much better prognosis. Since the term tuberculous infants is used for both types of patients and since the recent literature more frequently deals with the subclinical type, the impression of an improving prognosis for tuberculosis in infants is created.

However, even if one allows for the difference in the type of the tuberculous patients with which the recent literature deals, the course of tuberculous infection in children at present appears to be mild more frequently than in the past. It is probable that because of improved technic in diagnosis more persons who are sources of infection are recognized early now than in former years. Early diagnosis involves early precautions and measures for the prevention of the spread of the disease. It is reasonable to assume that fewer infants than formerly are now exposed to massive doses of tubercle bacilli.

It may be inferred, therefore, that the milder tuberculous infections and the greater number of early and subclinical diagnoses among infants are responsible for the apparently lower rate of deaths from tuberculosis among infants and children reported in the recent medical literature. If one could compare strictly analogous types of tuberculous infants of half a century ago and of the present, the difference in

prognosis might not be as great as it seems. No specific therapeutic measure applicable to infants which could account for the decrease in their death rate has been discovered.

As a part of the investigation of B C G which was started in December 1926 in New York, more than 1,000 nonvaccinated infants exposed to tuberculosis by contact have been observed as controls for from one to nine years. These infants may be divided into three groups: (1) infants known to us since the time of their birth; (2) infants not known to us since birth who gave a negative reaction to the tuberculin test at the time of their admission to the study, and (3) infants known to us for less than their whole lives who gave a positive reaction to the tuberculin test when we first saw them. Only the first two of these groups were used as controls in the B C G study, and the children of the third group were observed merely as an especially interesting series. Approximately 20 per cent of the infants used as controls were found to have become infected before the fifth year of life. This report deals with that 20 per cent, together with the children in the third category, i. e., infants already infected when first seen by us.

No attempt will be made in this report to study the frequency of infection or clinical tuberculosis among infants exposed by contact. We shall limit ourselves to a group of infected infants selected from more than 1,000 exposed infants. The difficulty of interpreting abnormal shadows in the roentgenograms of young children was previously pointed out by one of us, who with Park and Logie [1] showed that in a large series of films read by an expert roentgenologist who was not familiar with the clinical history of the patients or with their reactions to the tuberculin test one third of the mediastinal and one fifth of the parenchymal shadows described as abnormal occurred in apparently well children who showed a negative response to the tuberculin test.

The entire group of children considered in this report numbers 348. A summary of the group is presented in table 1. Of this total, 162 (46 per cent) were known from birth or showed a negative reaction to the tuberculin test at the time of admission to the study. The remaining 54 per cent were already infected when first encountered. The period during which individual children were studied ranged from one to nine years. Seventy-one per cent of the patients have been observed for longer than three years. The rate of deaths from tuberculosis for the entire group is 9.1 per cent. Two hundred and seventy-eight (79 per cent) of the group were infected during the first two years of life. Since no deaths occurred among the children who were infected after the

1. Kereszturi, C.; Park, William H., and Logie, A. J.: Nat. Tuberc. A. Tr. **31**:97, 1935.

eighteenth month of life, the rate of mortality from tuberculosis among the 278 children infected during the first two years of life is 11.5 per cent. Of the patients for whom roentgenograms were made, 28.9 per cent showed parenchymal shadows. However, only a few tuberculous children were clinically ill.

A subdivision of the patients as to race reveals that the rate of mortality from tuberculosis among the Negro children was approximately twice that of the white, the figures being 15.8 per cent and 7.1 per cent, respectively. The Puerto Rican children were about midway between the other two groups, with a death rate of 12.8 per cent. Similarly, more of the Negro children than of the white had parenchymal lesions, 44.1 per cent and 24.4 per cent, respectively, with the Puerto-Rican group again between the others.

TABLE 1.—*Tuberculous Infection During the First Five Years of Life*

Race of Patients	Number of Patients	Number of Deaths from Tuberculosis	Percentage of Patients				
			Of Whom Roentgenograms Were Taken	In Fourth Column Who Had Parenchymal Lesions	Exposed to Sputum Positive for Tuberculosis	Exposed to Sputum Negative for Tuberculosis	Not Known to Have Been Exposed
White.....	252	18 (7.1%)	50.4	24.4	83	13	4
Negro.....	57	9 (15.8%)	59.6	44.1	89	9	2
Puerto Ricans..	39	5 (12.8%)	48.7	31.6	88	6	6
Total.....	348	32 (9.1%)	51.7	28.9	85	11	4

All the patients were members of tuberculous families and became infected: Eighty-five per cent were known to have been exposed to open tuberculosis and 11 per cent to closed tuberculosis, while 4 per cent were not known to have been in actual contact with the tuberculous source (table 1).

The relationship of the age when infection occurred to the mortality from tuberculosis is presented in table 2. More than 40 per cent of those infected during the first three months of life died of tuberculosis. During the successive three month periods of the first year, the death rate gradually declines. It is interesting to note that no deaths from tuberculosis occurred in the group of 114 children who were infected at from 18 to 59 months of age, inclusive. The rate of mortality from tuberculosis of those infected when less than 1 year old was 14.8 per cent; those infected when 1 year of age or older had a mortality rate of 3.4 per cent. The importance of preventing infection early in life needs no further elucidation than these figures (table 2).

As was previously mentioned, this investigation has been conducted for more than nine years. An analysis of the group according to age,

as of Feb. 1, 1936, is presented in table 3. All the deaths from tuberculosis occurred in children less than 3 years of age. The rates were 10.3 per cent in the first, 3.5 per cent in the second and 1.8 per cent in the third year group (table 3).

TABLE 2.—*Relationship Between the Age at the Time of Tuberculous Infection and the Death Rate*

Age When Infected, Months	Number of Patients	Number of Deaths from Tuberculosis	Age When Infected, Years	Number of Patients	Number of Deaths from Tuberculosis
Less than 3	16	7 (43.7%)	Less than 1	175	26 (14.8%)
3-5	35	7 (20.0%)			
6-8	63	9 (14.3%)			
9-12	61	3 (4.9%)			
12-17	59	6 (10.2%)*	1-4	173	6 (3.4%)
18-23	44	0			
24-59	70	0			

* This death rate is higher than one would expect from the general trend of the figures. The explanation for it is that our study includes a smaller proportion of deaths from tuberculosis among the younger children than presumably would be the case had we limited our group to children known to us from their birth, instead of including children who had survived for varying periods before coming under our observation. In those patients known to us since birth, the death rate at the age of from 12 to 17 months was lower than during the preceding six month period.

TABLE 3.—*Rate of Deaths from Tuberculosis in Relation to Age Groups*

Age, Years	Number of Patients	Number of Deaths from Tuberculosis
Less than 1	175	18 (10.3%)
More than 1	256	9 (3.5%)
More than 2	283	5 (1.8%)
More than 3	248	.....
More than 4	202	.....
More than 5	159	.....
More than 6	139	.....
More than 7	108	.....
More than 8	77	.....
More than 9	34	.....

TABLE 4.—*Distribution of Deaths According to Age*

Total Number of Deaths from Tuberculosis	Age, Years		
	First Year	Second Year	Third Year
32	18 (56.2%)	9 (28.1%)	5 (15.7%)

The distribution of deaths according to age is seen in table 4 and reveals that 56.2 per cent of the deaths occurred in the first year, 28.1 per cent in the second and 15.7 per cent in the third.

A study of the roentgenograms of the chest of the children infected during the first two years of life and the relationship of the roentgeno-

graphic observations to the mortality from tuberculosis are presented in table 5. As was to be expected, the highest mortality occurred in the group who had visible parenchymal lesions. The mortality rate in that group was approximately five times as great as among those children whose roentgenograms showed enlarged tracheobronchial glands and was about eight times as great as in those children who gave no roentgenographic evidence of tuberculosis. The rate of mortality among the Negro children with parenchymal shadows was almost twice that of the white. The Puerto-Rican children whose lungs presented a pathologic aspect in the roentgenograms numbered only 5, of whom 4 died (table 5).

TABLE 5.—*Relationship of Rate of Deaths from Tuberculosis to the Roentgenographic Observations on Children Infected During the First Two Years of Life*

Roentgenographic Observations	White		Negro		Puerto Rican		Totals	
	Number of Patients	Deaths from Tuberculosis	Number of Patients	Deaths from Tuberculosis	Number of Patients	Deaths from Tuberculosis	Number of Patients	Deaths from Tuberculosis
Abnormal parenchymal shadows	27	7 (25.9%)	15	7 (46.7%)	5	4 (80%)	47	18 (38.3%)
Enlarged tracheobronchial glands	11	1 (9.1%)	2	..	..	..	13	1 (7.7%)
No evidence of tuberculosis	65	4 (6.2%)	14	..	8	..	87	4 (4.5%)
No roentgenogram taken	97	6 (6.2%)	19	2 (10.5%)	15	1 (6.7%)	131	9 (6.8%)
Total.....	200	18 (9.0%)	50	9 (18%)	28	5 (17.8%)	278	32 (11.5%)

COMMENT

G. G. Kayne [2] in 1935 made an extensive survey of the literature concerning the fate of tuberculous infants. However, the extreme variations in death rate between the 1.7 per cent recorded by G. Lissant Cox [3] and the 62 per cent given by Asserson [4] mean little if one analyzes the different groups of patients that various authors have described under similar headings. Some writers, like Dow and Lloyd [5] and Cox,[3] studied infants exposed to tuberculosis by contact and did not exclude those who continued to give a negative reaction to the tuberculin test. Such groups naturally have a good prognosis. On the other

2. Kayne, G. G.: Brit. M. J. **1**:692, 1935.

3. Cox, G. Lissant, and others: Tubercle **101**:497, 1929.

4. Asserson, M. A.: Am. Rev. Tuberc. **16**:359, 1927.

5. Dow, D. F., and Lloyd, W. E.: An Investigation from the Department of the Brompton Hospital for Consumption and Diseases of the Chest, no. 1, Bungay, Suffolk, Chaucer Press.

hand, Asserson [4] selected a group of infants who showed a positive reaction to the tuberculin test and some of whom had marked clinical signs and symptoms of tuberculosis. Such a group would be expected to have a poor prognosis.

In addition to such differences in the patients studied by various investigators, there are differences in the type and the length of exposure to tuberculosis, which few authors specify. These factors are vitally important in estimating results. Debré, Lelong and Bernard [6] made an interesting comparative study of the significance of exposure to tuberculosis. They found that, of 265 infants exposed to tuberculosis who were separated from the tuberculous source before they became infected, none died of tuberculosis. Among 171 patients who were separated from the source after they became infected but while they were in good condition and had no clinical disease, 7.6 per cent died of tuberculosis. Finally, of 66 patients who became infected and remained in the same environment, 82 per cent died in the course of four years during which they were observed.

A statistical comparison of figures originating in different countries, or even in different communities in the same country, is somewhat hazardous. The incidence of tuberculosis, the type of population—urban or rural—and the economic status of the people might be significantly different.

In New York we were able to find but two extensive studies on the subject. One was published by McLean and Jeidell [7] in 1922. They obtained their material from the old Babies Hospital and found a death rate of 51 per cent among infants known to be tuberculous in the first year of life, of 33 per cent among those who were observed to be infected during the second year and of 23.3 per cent among those in their third year when their condition was diagnosed as tuberculous. Our figures are computed for deaths from tuberculosis exclusively, and they show a death rate of 14.8 per cent for those infected during the first year and of 10.2 per cent for those whose infection occurred when they were at the age of from 12 to 17 months. We observed no deaths from tuberculosis among the 114 patients infected after reaching 18 months of age.

The other extensive study in New York was made by Asserson [4] and published in 1927. She made a survey of children of from 1 to 5 years of age and in the care of different hospitals and found the rate of deaths from tuberculosis to be 82 per cent among children who became clinically tuberculous with parenchymal lesions of the lungs within the first two years of life. A similar group of 47 patients in our study had

6. Debré, R.; Lelong, M., and Bernard, L.: Presse méd. **33**:345, 1925.

7. McLean, S., and Jeidell, H.: Pirquet Tests, Am. J. Dis. Child. **24**:73 (July) 1922.

a death rate of only 38.3 per cent. Asserson found the rate of deaths from tuberculosis to be 3 per cent among children who showed no roentgenographic evidence of tuberculosis or who presented only a lesion limited to the tracheobronchial lymph nodes. In our study there were 100 such patients, with a rate of deaths from tuberculosis of 5 per cent.

Drolet [8] found ten times more deaths from tuberculosis among Negro children under 15 years of age than among white ones during the years 1930-1932. Our experience in the present study shows that Negro patients have only about twice as high a rate of deaths from tuberculosis as have white patients. However, we are dealing only with infected patients. Drolet's figures are for children from the general population, and the incidence of infection among Negro children in New York is somewhat higher than among white children.

Osswald [9] in 1922 made an observation similar to ours, namely, that all the deaths from tuberculosis among infected infants occur in the first three years of life. Our results both as to the mortality of tuberculosis and the racial differences closely approximate those recently reported by Brailey [10] of the Johns Hopkins University.

We feel that our material in this paper fairly represents the fate of infected children of tuberculous parents for the following reasons:

1. About half of the patients were known to us before they became infected.

2. Tuberculin tests were periodically repeated.

3. Roentgenograms of the chest were taken for about half of the patients.

4. The study covered observations of individual children for from one to nine years, and 71 per cent of the patients were observed for longer than three years.

5. In about half of the cases in which the patient died autopsy examination was made.

SUMMARY

1. In a group of 348 infants infected with tuberculosis during the first five years of life and observed for from one to nine years the rate of deaths from tuberculosis was 9.1 per cent. Of the patients for whom roentgenograms of the chest were taken, 28.9 per cent presented parenchymal lesions of the lungs.

2. The rate of mortality from tuberculosis among the Negro children was approximately twice that among the white ones. The incidence of parenchymal lesions of the lungs also was higher in the Negro group.

8. Drolet, G. J.: Am. Rev. Tuberc. **30**:1, 1934.

9. Osswald, R.: Monatschr. f. Kinderh. **23**:168, 1922.

10. Brailey, Miriam E.: Bull. Johns Hopkins Hosp. **59**:1, 1936.

3. The rate of deaths from tuberculosis among 175 children infected with the disease during the first year of life was 14.8 per cent; the 59 who were infected during the first half of the second year showed a rate of 10.2 per cent. The highest rate—43.7 per cent—occurred in the children infected during the first three months of life.

4. No deaths from tuberculosis occurred in the group of 114 children infected after reaching 18 months of age.

5. All the deaths from tuberculosis occurred in children less than 3 years of age.

6. The rate of deaths from tuberculosis was more than 5 times as high in the children with parenchymal lesions as in those with enlarged tracheobronchial glands and about eight times as high as in those who gave no roentgenographic evidence of having tuberculosis.

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RESULTS OF BLOOD TRANSFUSIONS IN PRIMARY PNEUMONIA IN INFANTS AND IN CHILDREN

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The intravenous administration of any appreciable quantity of fluid in cases of pneumonia has usually been considered a hazardous procedure because it increases the load of an already overtaxed myocardium and pulmonary circulation. However, there is little ground for the belief that the circulation will be embarrassed by transfusions at a time when the total circulating volume is decreased. To normal dogs with vascular beds that contained the normal quota of blood, Meek and Eyster [1] gave intravenous injections of whole blood, physiologic solution of sodium chloride and solution of acacia in quantities varying from 25 to 103 per cent of the total blood volume without increasing permanently the diastolic size of the heart as demonstrated by roentgen ray measurements. Estimations of the hemoglobin content indicated that most of the injected fluid was still in circulation; the capillaries and venules acted as reservoirs for the storage of excess fluid. Rose and Lund [2] reported that blood transfusions were of some value in cases of pneumonia complicating influenza. Bass [3] had remarkable results with this form of therapy in cases of pneumonia in anemic infants. Flinn [4] and Weston [5] described immediate recovery following transfusion in two infants with pneumonia and severe anemia. Carlton,[6] however, found little benefit from this form of therapy, but he did not mention any harmful effects, nor did he mention the presence of anemia in any of the patients.

In the past three years, efforts were made at the Duke Hospital to give one or more blood transfusions to all infants and children who had severe primary pneumonia, whether or not they had anemia. However, because of the difficulty in obtaining suitable donors, thirty-five of these

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1. Meek, W. J., and Eyster, J. A. E.: Am. J. Physiol. **61**:186, 1922.
2. Rose, C. W., and Lund, E. J.: Treatment of the Pneumonic Disturbance Complicating Influenza, J. A. M. A. **72**:642 (March 1) 1919.
3. Bass, M. H.: The Association of Anemia and Pneumonia in Infants, Am. J. Dis. Child. **29**:318 (March) 1925.
4. Flinn, L. B.: Blood Transfusion in Anemic Infants with Pneumonia, Am. J. Dis. Child. **37**:596 (March) 1929.
5. Weston, W.: Arch. Pediat. **44**:378, 1927.
6. Carlton, C. H.: Lancet **2**:850, 1926.

patients did not receive blood and consequently served as controls (table 1). The other twenty-four had one or more transfusions of citrated blood by the gravity method (maximal amount, 20 cc. per kilogram of body weight). The clinical symptoms, hemoglobin content and red and white blood cell count for the two groups were almost identical (tables

TABLE 1.—*Results of Blood Transfusion in Cases of Primary Pneumonia in Infants and Children*

Total Number of Patients	Crisis or Lysis on Day of Transfusion	Crisis or Lysis From 24 to 48 Hours After Transfusion	Average Interval Between Onset and Crisis or Lysis, Days	Complications	Deaths
24 (received transfusions).....	15 (62.5%)	9 (37.5%)	7.9	4 (12%)	1 (4%)
35 (did not receive transfusions)	.....	.....	9.9	12 (34%)	5 (14%)

TABLE 2.—*Age, Hemoglobin Content and Red and White Blood Cell Count on Admission to the Duke Hospital of Patients Who Received Transfusions*

Patient	Age		Hemoglobin Content, Percentage	Red Blood Cells	White Blood Cells
	Years	Months			
D. B.	3	..	70	.....	22,000
B. C.	2	..	75	4,230,000	33,450
G. B.	..	2	83	3,750,000	21,600
P. S.	3	..	80	3,632,000	32,400
G. A.	2	..	50	3,520,000	40,000
P. C.	..	10	55	3,092,000	14,000
J. A.	..	6	80	4,580,000	30,520
D. G.	..	16	95	4,480,000	11,760
B. W.	..	1	100	4,658,000	16,640
B. W.	..	14	55	3,630,000	43,000
M. S.	7½	..	75	3,650,000	20,500
L. R.	1	..	35	1,850,000	94,000
D. H.	2	..	80	4,372,000	17,320
J. P.	2	..	70	3,820,000	30,600
W. L.	..	9	72	3,610,000	51,300
T. M.	9	..	87	4,000,000	14,100
D. E.	1	..	45	3,500,000	19,920
J. S.	..	10	40	3,200,000	30,800
E. F.	7	..	62	3,120,000	18,200
N. R.	1½	..	70	3,350,000	43,600
F. K.	..	4	74	4,840,000	20,650
C. J.	2	..	75	4,190,000	17,200
K. S.	3	..	75	3,870,000	20,200
M. R.	4	..	77	4,020,000	18,750
Mean	2	..	75	3,750,000	20,650

2 and 3). The only difference which could be noted was that in the group who received transfusions there were probably more ill patients and more infants.

Prompt and marked symptomatic improvement usually followed the transfusion, and the patient appeared more comfortable. The temperature fell by crisis within twenty-four hours of the transfusion in fifteen patients and within forty-eight hours in the other nine (charts 1, 2 and 3). The average interval between the onset and the crisis or lysis

for this group was seven and nine-tenths days. Four of these children had the complication of suppurative otitis media, and one died. For the thirty-five patients who did not receive transfusions, the average interval between the onset and the crisis or lysis was nine and nine-tenths days. Three of these children had the complication of empyema, and nine had otitis media. Five died.

The severe dyspnea and cyanosis which many of these children had were improved instead of made worse by the transfusions. No circu-

TABLE 3.—*Age, Hemoglobin Content and Red and White Blood Cell Count on Admission to the Duke Hospital of Patients Who Did Not Receive Transfusions*

Patient	Age		Hemoglobin Content, Percentage	Red Blood Cells	White Blood Cells
	Years	Months			
A. N.	7	..	90	5,180,000	36,000
P. C.	..	17	52	3,806,000	11,000
D. B.	..	6	80	4,200,000	12,400
M. P.	8	..	..	4,300,000	51,250
W. B.	..	7	55	3,920,000	8,080
F. M.	7	..	62	4,760,000	20,000
L. C.	..	3	80	3,710,000	18,450
W. W.	6	..	70	4,660,000	30,200
J. W.	2	..	77	3,740,000	22,560
B. S.	..	19	53	3,910,000	24,950
C. T.	4	..	79	3,700,000	54,950
V. L.	..	14	70	3,800,000	14,400
C. F.	14	..	75	3,570,000	11,600
J. C.	4	..	74	3,900,000	20,400
E. D.	11	..	89	3,900,000	12,200
L. M.	..	14	74	3,710,000	12,800
H. G.	12	..	95	3,570,000	23,800
E. G.	2	..	74	3,510,000	17,200
C. N.	6	..	83	4,500,000	19,640
T. F.	4	..	66	3,630,000	14,560
D. D.	10	..	80	.....	27,040
M. J.	6	..	60	2,510,000	27,840
H. D.	6	..	90	4,010,000	21,650
J. A.	5	..	76	3,860,000	40,000
C. W.	13	..	95	4,250,000	21,000
J. D.	3½	..	61	4,630,000	28,800
J. H.	12	..	78	3,660,000	18,950
C. Y.	..	5	97	.....	28,800
G. B.	4	..	66	4,610,000	20,300
L. S.	3	..	80	3,960,000	26,750
S. C.	..	3	75	3,740,000	33,950
D. D.	7	..	80	4,300,000	22,000
B. C.	2	..	55	2,980,000	26,200
J. C.	5	..	90	4,800,000	14,000
R. T.	8	..	85	4,350,000	17,800
Mean	4	..	75	3,800,000	21,000

latory embarrassment was noted during or after the procedure. After the transfusion several of the patients had an immediate rise in temperature, which was usually followed by an immediate crisis (chart 3).

No explanation for the beneficial effect of the blood transfusions is known. That the correction of anemia was a factor seems doubtful.[7]

7. (*a*) Lyon, A. B.: Boston M. & S. J. **185**:184, 1921. (*b*) Kugelmass, I. N., and Lampe, M.: The Mechanism of Anemia Associated with Infection in Infancy and in Childhood, Am. J. Dis. Child. **43**:291 (Feb.) 1932. (*c*) Leake, C. D., and Brown, T. K.: J. Exper. Med. **36**:661, 1922.

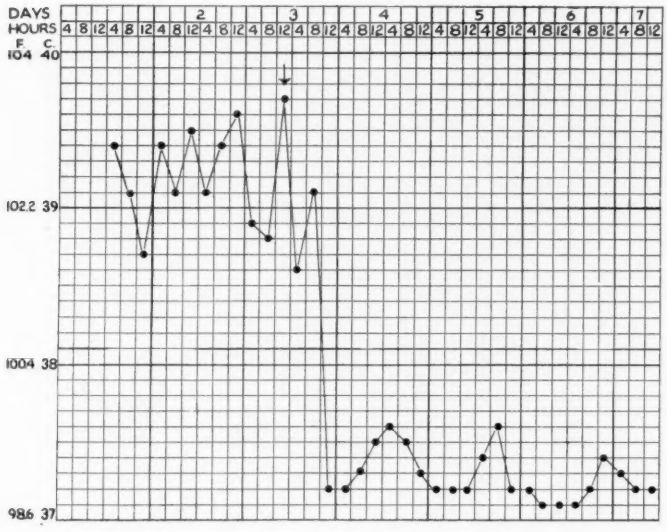

Chart 1.—Temperature chart of L. R. The arrow indicates a transfusion on the third day at the hospital.

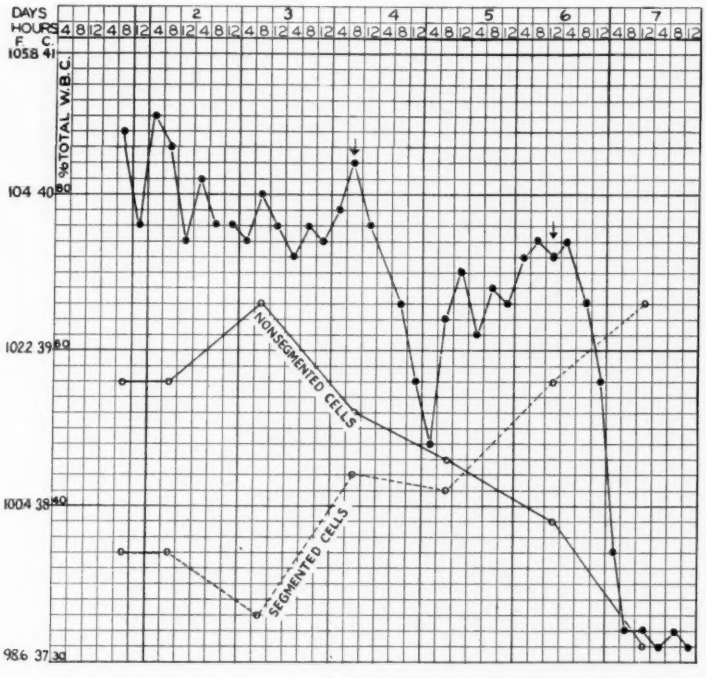

Chart 2.—Temperature chart of T. M. The arrows indicate a transfusion on the fourth and on the sixth day at the hospital. The ratio of segmented to non-segmented cells as the crisis approached is noteworthy.

The results were as good in the patients with severe anemia as in those with moderate anemia. Of the five children in the control series who died, only two had a hemoglobin content below 70 per cent (10.8 Gm. per hundred cubic centimeters). Possibly, transfusions stimulate an increase in segmented polymorphonuclear leukocytes. The hemogram studies which have been made for several patients with primary pneumonia indicate that improvement or a crisis is attended by an increase

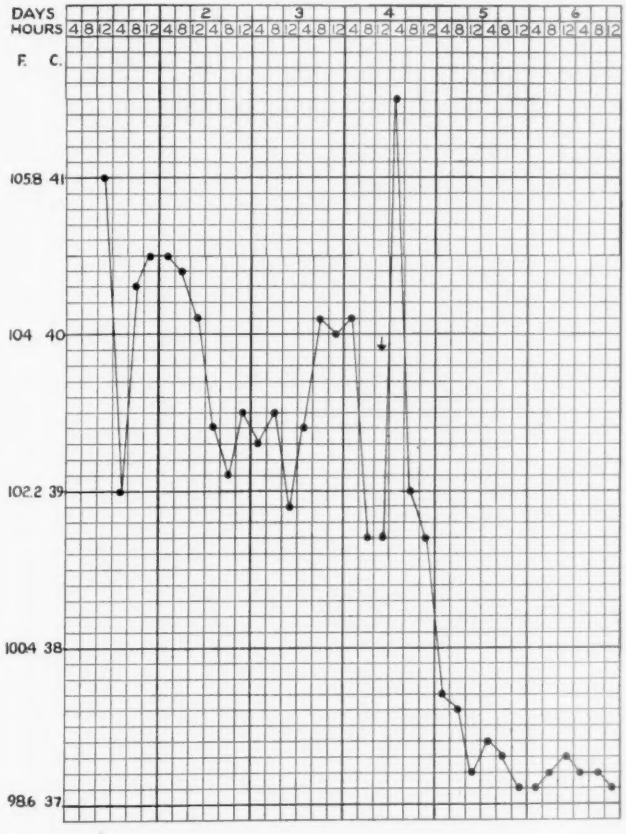

Chart 3.—Temperature chart of M. S. The arrow indicates a transfusion on the fourth day at the hospital. The reaction to the transfusion was followed by a prompt crisis.

in the segmented polymorphonuclear leukocytes, with a decrease in the percentage of nonsegmented forms (chart 2). Although these studies have been few, the ratio of segmented to nonsegmented forms appears to be a good indication of the severity of the infection and the ultimate outcome. Perhaps transfusions are a form of serotherapy, introducing neutralizing antibodies or causing nonspecific protein shock. By the

increase in the blood volume, the heart may be so affected as to overcome the cardiac weakness. Another factor may be the supplying of cells to carry needed oxygen to the tissues, the replacing of those withdrawn from the circulation by the pathologic process.[7e]

SUMMARY

1. Heretofore pneumonia has been considered a contraindication for blood transfusion, but in our experience the chances for recovery seem to be increased by its use. Apparently the only contraindication for transfusions in children is damage to the kidneys, and if blood which causes any sign of hemolysis is excluded, even children with that condition can be given citrated blood intravenously.[8]

2. A crisis occurred within twenty-four hours of the transfusion in 62.5 per cent of the patients and within forty-eight hours in the remainder. The average interval between the onset and the crisis was shorter and the mortality lower for the children who received transfusions than for those who did not.

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8. Bordley, J.: Reactions Following Transfusion of Blood with Urinary Suppression and Uremia, Arch. Int. Med. **47**:288 (Feb.) 1931. Hansen-Pruss, O. C. E.: To be published.

CALORIC AND PROTEIN REQUIREMENTS AND BASAL METABOLISM OF CHILDREN FROM FOUR TO FOURTEEN YEARS OLD

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DETROIT

The existing data on the food requirements of children were reviewed and summarized by the Committee on Growth and Development of the White House Conference on Child Health and Protection in 1932.[1] Since then two reports[2] have appeared on American girls from 6 to 16 years of age and a third[3] on a group of 206 children of both sexes from 2 to 11 years old. The present report on 14 boys and 13 girls is placed on record as representing comparatively long periods of observation (averaging about nine months), with parallel observations of the basal metabolism and the nitrogen balance and with variations in the protein intake, the other variables in the food mixture being controlled. The study extended over four years and included 56 children, the records for 29 of whom were excluded from this report because of infections or abnormalities in metabolism or nutrition.

PLAN AND METHODS OF STUDY

It was our plan to start the subjects on measured diets at caloric levels slightly lower than those at which gain in weight and height might be expected and to increase the caloric intake by degrees until there were gain in weight and height, a positive nitrogen balance and satisfaction of appetite. With the caloric intake constant, the protein content of the diet was then varied in an attempt to determine the optimal protein intake. While we are not prepared to answer the question as

From the Department of Pediatrics and the Department of Laboratories, Henry Ford Hospital.

1. Growth and Development of the Child: III. Nutrition, White House Conference on Child Health and Protection, New York, Century Company, 1932.

2. Wait, B., and Roberts, L. J.: Studies in Food Requirement of Adolescent Girls: Protein Intake of Well-Nourished Girls Ten to Sixteen Years of Age, J. Am. Dietet. A. **8**:403 (Jan.) 1933. Koehne, M., and Morrell, E.: Food Requirement of Girls from Six to Thirteen Years of Age, Am. J. Dis. Child. **47**:548 (March) 1934.

3. Lucas, W. P.; Pryor, H. B., and Pope, S. T., Jr.: Analysis of Sample Diets and Daily Habits of Two Hundred and Six Children from Data Supplied by Parents, J. Pediat. **4**:221 (Feb.) 1934.

to the criteria for an optimal diet, it seems to us that on that diet the subject should manifest, in addition to the items already mentioned (gain in weight and height, a positive nitrogen balance and satisfaction of appetite), a normal basal metabolism. In another article [4] we pointed out the adjustment which the basal metabolic rate is observed to make to a submaintenance diet. So conservative a measure is this that superficially it might seem possible to meet the requirements for growth with lower levels of intake than are conventional, but this would be found to have taken place at the expense of the basal metabolic rate. This point was discussed at length by Benedict,[5] who found disturbances of blood composition, of normal sex expression and of neuromuscular efficiency and the appearance of mental and physical unrest coexistent with the lowering of the metabolic rate produced by long periods of caloric curtailment. Accordingly, we have added a normal basal metabolic rate as a further requirement to be satisfied in selecting the number of calories and the amount of protein for the diet of any given person.

The children were hospitalized as reactors to tuberculin whose homes had been broken up because of disease in the parents, but in the report data are not included for any child who himself showed any active disease. The children attended school in the hospital grounds; they played outdoors when the weather permitted, and in season, in place of attending school, worked in their own gardens. Exclusion of many children from the report was necessary on the basis of their marked variation from the average in weight or in basal metabolism; intercurrent illness; lack of cooperation, or premature discharge from the hospital

The diets used were calculated from the revised food standards of Atwater and Bryant [6] and Waller.[7] Fifty sample diets, representing a total day's intake, were digested with sulfuric acid and analyzed for nitrogen by the Kjeldahl-Gunning procedure. The average values thus obtained exceeded by 11.75 per cent the calculated nitrogen value. This correction was used in calculating the nitrogen balances.

In studies of the caloric requirement, the calories in the diet were apportioned 15 per cent to protein, 35 per cent to fat and 50 per cent to carbohydrate. This percentage composition was that noted by Holt and Fales,[8] though so far as we know there exist no data to justify its being considered optimal. One point in its favor, however—aside from its being the one apparently self-selected by their subjects—is that when it is given the basal metabolic mixture usually reverses these proportions of fat and carbohydrate (making them 50 per cent fat and 35 per cent carbohydrate), and a basal oxidation of a percentage of carbohydrate much lower than that would approach a ketogenic mixture.

When the percentage of protein was varied, the change in calories of fat and of carbohydrate was apportioned equally, the total number of calories being

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4. Johnston, J. A., and Maroney, J. W.: Relationship of Basal Metabolism to Dietary Intake, Am. J. Dis. Child. **51**:1039 (May) 1936.

5. Benedict, F. G.; Miles, W. R.; Roth, P., and Smith, H. M.: Human Vitality and Efficiency Under Prolonged Restricted Diet, Washington, D. C., Carnegie Institution of Washington, 1919.

6. Atwater, W. O., and Bryant, A. P.: Chemical Composition of American Food Materials, Bulletin 26, United States Department of Agriculture, 1906.

7. Waller, D. S.: Nutritive Value of Food, Ann Arbor, Mich., George Wahr, 1932.

8. Holt, L. E., and Fales, H. L.: Food Requirements of Children: Percentage Distribution of Calories, Am. J. Dis. Child. **24**:311 (Oct.) 1922.

kept constant. The protein of the diet was derived approximately 65 per cent from animal and 35 per cent from vegetable sources. The diets were varied and were made as attractive as possible within the limits of the caloric specifications. The children were encouraged to eat whatever was included in the diets. A spirit of competition in making perfect records for eating everything served was fostered, and excellent cooperation was obtained. Any part of the diets refused was weighed, and its nitrogen and caloric value was estimated. Children who did not cooperate or who refused more than 5 per cent of the diet were excluded from the report.

Spotted three day collections of urine and stool, or in some instances continuous collections of urine and stool, were made. In general, determinations of nitrogen in the urine and stool were made for nine or more days with each change in diet. Collections of urine and stool were analyzed for nitrogen by the Kjeldahl-Gunning procedure. Observations on basal metabolism were made to parallel changes in diet. Collections of gas were made with the Tissot tank, and analyses, with the Haldane-Henderson apparatus.

The 27 children reported on here were kept on measured diets for periods averaging nine and four-tenths months, and determinations of nitrogen in the urine and stool were made for an average of one hundred and eleven days for each child.

FINDINGS

The findings are recorded in detail in five tables and five charts—one table and one chart for each of five subjects the observations on whom were representative of those on the whole group. The findings for the entire group appear in two additional tables.

In the charts are shown the periods during which the diets were used; the weight curve (based on smoothed line curves of daily weights); the estimated gain in weight calculated from the Baldwin-Wood standards; the nitrogen balances, with the solid black bars representing three day balances and the shaded areas representing the averages of the three day balances in each period; the caloric intake, and the basal calories. The data from which each chart was prepared are shown in one of the tables.

SUBJECT 1.—M. G. (chart 1 and table 1), a Negro boy, was studied from the age of 6 years and 7 months through the age of 7 years and 8 months.

In the first four periods, from March 27 to August 19, the diet was increased from 900 calories to 1,500 calories, with the protein kept at 15 per cent of the total intake. With 900 calories there were loss of weight and a negative nitrogen balance; with 1,100 calories, though the nitrogen balance became positive, gain in weight was only average, and appetite was not satisfied, but with either 1,250 or 1,400 calories there were both gain in weight and a positive nitrogen balance. In the three periods from August 20 to October 30 the intake was increased to 1,650 calories, and the percentage of protein varied from 15 to 10 to 20 per cent, with storage of nitrogen and gain in weight at each level, the total amount of food, however, proving more than the child's appetite demanded. In the next four periods, from November 2 to January 30, the intake was reduced to 1,500 calories, and the protein varied from 15 to 20 to 25 to 10 per cent. There were

positive nitrogen balances with 15, 20 and 25 per cent of protein; with 10 per cent of protein a negative balance was obtained. The negative nitrogen balance in this instance might be attributed to an unloading of the high retention in the previous period; however, a majority of our subjects showed negative balances when the percentage of protein was reduced to 10 per cent, on total caloric intakes sufficient to produce storage of nitrogen and gain in weight when the percentage

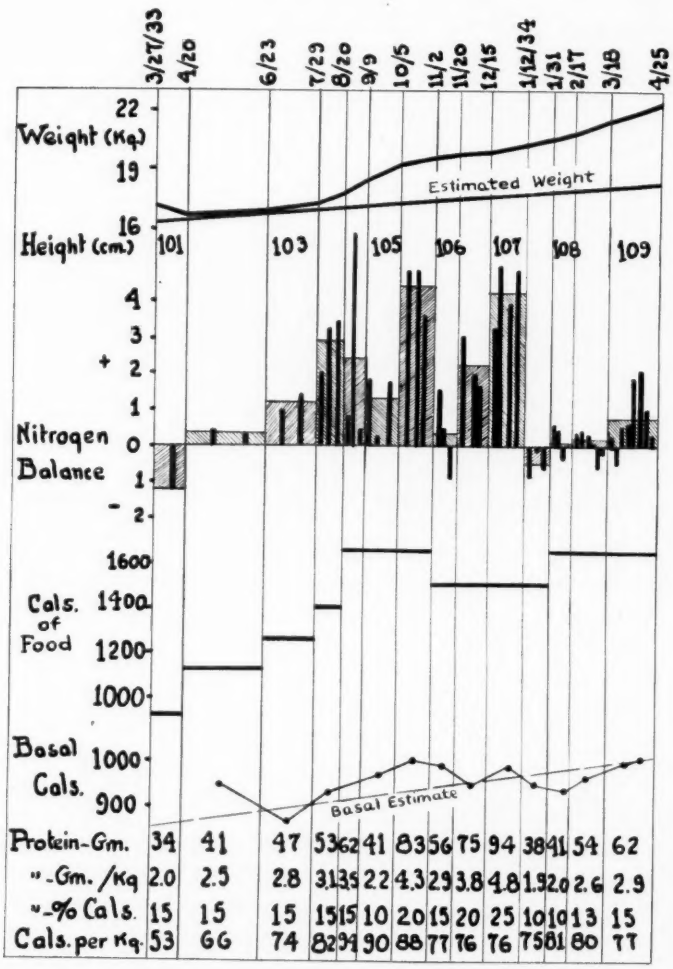

Chart 1.—Results of the study of subject M. G. In this chart and in charts 2, 3 and 4, the solid black bars represent three day nitrogen balances, and the shaded areas, the averages of the three day balances in each period. The "estimated weights" in the same four charts were calculated according to age and height from the Baldwin-Wood tables of predicted gains in weight. The "basal estimates" in all the charts were taken from the Boothby-Sandiford tables of predicted basal metabolic rates.

TABLE 1.—*Observations on M. G. (Male, Born on Aug. 15, 1927)*

TABLE 1.—*Observations on M. G. (Male, Born on Aug. 15, 1927)*

Period of Diet	Dates of Three Day Collections	Calories of Food	Calories per Kg. of Weight	Protein, Percentage of Calories	Protein, Gm.	Protein, Gm. per Kg. of Weight	Protein Nitrogen	Nitrogen in Urine	Nitrogen in Stool	Nitrogen Balance	Basal Calories	Basal Calories per Unit of Surface Area	Basal Metabolic Rate	Respiratory Quotient, Percentage	Weight, Kg.	Estimated Weight, Kg.	Height, M.	Age, Yr.-Mo.
3/27/33	4/12-14	900	53	15	33.6	2.0	6.01	6.48	0.80	—1.27	...	....	....	..	17.0	16.4	1.01	5-8
4/19																		
4/20	5/14-17	1,100	66	15	41.2	2.5	7.38	6.13	0.87	+0.38	...	....	....	..	16.6			
6/22	5/27-29	.....	..	..	....	...	.....	5.74	1.35	+0.29	950	58.2	+10	78				
6/23	7/10-12	1,250	74	15	46.9	2.8	8.38	6.44	0.99	+0.95	876	53.7	+ 1	84	16.8	....	1.03	
7/28	7/17-19	.....	..	..	....	...	.....	6.24	0.71	+1.43								
7/29	8/ 4-6	1,400	82	15	52.5	2.1	9.39	6.30	1.09	+2.00	...	....	....	..	17.1			
	8/11-13	.....	..	..	....	...	.....	5.24	0.89	+3.26	939	56.6	+ 7	83				
8/19	8/16-19	.....	..	..	....	...	.....	5.29	0.62	+3.48								
8/20	8/25-27	1,650	94	15	61.9	3.5	11.06	9.16	1.05	+0.85	...	....	....	..	17.5			
	8/30-9/1	.....	..	..	....	...	.....	4.14	0.98	+5.94								
9/ 8	9/ 6-8	.....	..	..	....	...	.....	9.17	1.44	+0.45								
9/ 9	9/17-20	1,650	90	10	41.3	2.2	7.38	4.19	1.30	+1.89	972	56.2	+ 6	83	18.4	....	1.05	6-1
	9/24-26	.....	..	..	....	...	.....	4.12	1.49	+0.25								
10/ 4	9/29-31	.....	..	..	....	...	.....	5.24	1.89	+1.77								
10/ 5	10/ 9-12	1,650	88	20	82.5	4.3	14.75	10.47	0.71	+4.87	...	....	....	..	19.0			
	10/21-23	.....	..	..	....	...	.....	9.02	0.88	+4.85	1,006	58.2	+10	83				
10/30	10/25-27	.....	..	..	....	...	.....	8.53	1.35	+3.57								
11/ 2	11/ 7-9	1,500	77	15	56.3	2.9	10.06	7.47	1.00	+1.59	...	....	....	..	19.4	....	1.06	
	11/10-12	.....	..	..	....	...	.....	8.10	1.46	+0.50	994	55.0	+ 4	81				
11/19	11/14-16	.....	..	..	....	...	.....	9.87	1.12	—0.93								
11/20	11/27 29	1,500	76	20	75.0	3.8	13.41	8.66	1.70	+3.05	...	....	....	..	19.7	....	....	6-3
	12/ 4-7	.....	..	..	....	...	.....	10.55	0.90	+1.96								
12/ 4	12/ 8-10	.....	..	..	....	...	.....	10.42	1.35	+1.64	950	52.9	+ 0	82				
12/15	12/18-20	1,500	76	25	93.8	4.8	16.76	11.67	1.89	+3.20	...	....	....	..	19.7	....	1.07	
	12/21-22	.....	..	..	....	...	.....	10.97	0.89	+4.90								
	12/29-31	.....	..	..	....	...	.....	11.41	1.42	+3.93	986	54.8	+ 3	84				
1/11/34	1/ 5-7	.....	..	..	....	...	.....	9.60	2.31	+4.85								
1/12	1/16-18	1,500	75	10	37.5	1.9	6.70	6.47	1.12	—0.89	...	....	....	..	20.0	....	....	6-5
	1/20-22	.....	..	..	....	...	.....	5.49	1.25	—0.04								
1/30	1/24-26	.....	..	..	....	...	.....	6.77	0.61	—0.68	948	52.8	+ 0	85				
1/31	2/ 4-6	1,650	81	10	41.3	2.0	7.38	6.08	0.69	+0.61	...	....	....	..	20.2	....	1.08	
	2/ 7-9	.....	..	..	....	...	.....	5.94	1.00	+0.46								
2/16	2/10-12	.....	..	..	....	...	.....	6.40	1.40	—0.42	936	51.6	— 3	84				
2/17	2/21-22	1,650	80	13	53.6	2.6	9.61	8.16	1.06	+0.39	...	....	....	..	20.6			
	2/26-28	.....	..	..	....	...	.....	6.55	1.61	+0.45								
	3/ 1-3	.....	..	..	....	...	.....	8.08	1.19	+0.34	964	52.3	— 1	78				
	3/ 4-6	.....	..	..	....	...	.....	7.84	1.75	+0.02								
	3/ 7-9	.....	..	..	....	...	.....	9.09	1.10	—0.58								
3/17	3/10-12	.....	..	..	....	...	.....	8.50	1.43	—0.22								
3/18	3/19-21	1,650	77	15	61.9	2.9	11.06	9.36	1.44	+0.26	...	....	....	..	21.4			
	3/23-25	.....	..	..	....	...	.....	9.93	1.77	—0.64								
	3/27-28	.....	..	..	....	...	.....	9.46	1.07	+0.53	995	52.6	— 1	86				
	4/ 5-7	.....	..	..	....	...	.....	9.45	0.99	+0.62								
	4/ 8-10	.....	..	..	....	...	.....	7.89	1.28	+1.89								
	4/14-16	.....	..	..	....	...	.....	7.54	1.45	+2.07								
	4/17-19	.....	..	..	....	...	.....	8.45	1.58	+1.03								
4/24	4/22-24	.....	..	..	....	...	.....	9.35	1.35	+0.36	1,006	52.9	+ 0	84	22.2	....	1.09	6-8

of protein was 15 or greater. In the last three periods, from January 31 to April 24, the intake was increased to 1,650 calories, and the protein was increased from 10 to 13 to 15 per cent. There was a positive nitrogen balance at all three levels. With an increase in the total intake, a positive nitrogen balance could frequently be obtained.

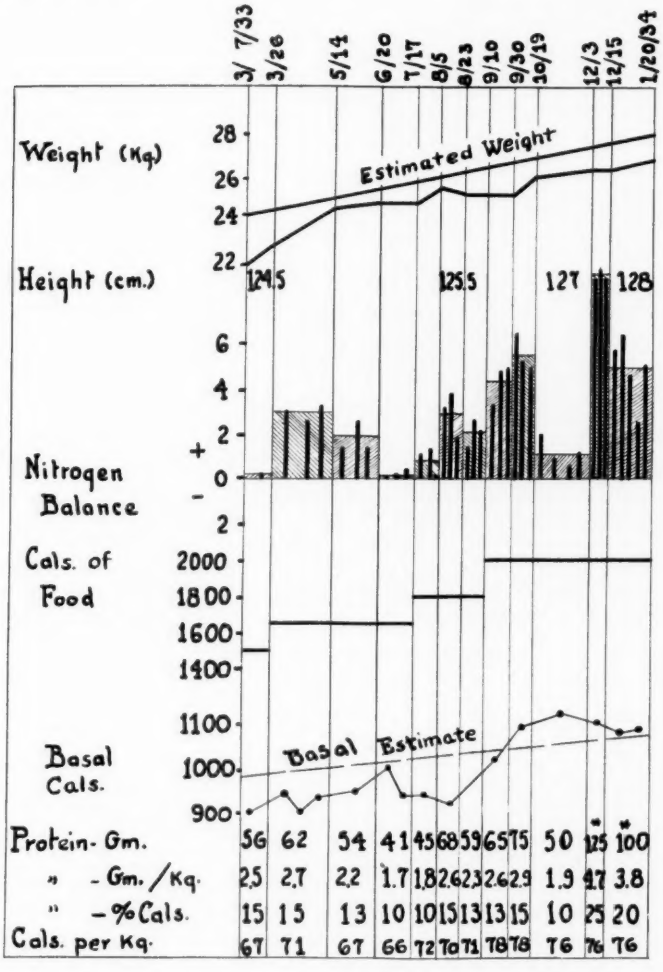

Chart 2.—Results of the study of subject E. M. Note that though gain in weight and a positive nitrogen balance were obtained with 1,650 calories while the child was recovering from malnutrition, subsequent sustained gain required 2,000 calories, apparently as a result of the rise in the basal metabolic rate.

In this subject the requirements of growth and storage of nitrogen were fulfilled on 1,250 calories with 15 per cent of protein and on 1,650 calories with 10 per cent of protein. Referred to weight, these values represent 74 calories with

TABLE 2.—*Observations on E. M. (Female, Born on April 16, 1923)*

Period of Diet	Dates of Three Day Collections	Calories of Food	Calories per Kg. of Weight	Protein, Percentage of Calories	Protein, Gm.	Protein, Gm. per Kg. of Weight	Protein Nitrogen	Nitrogen in Urine	Nitrogen in Stool	Nitrogen Balance	Basal Calories	Basal Calories per Unit of Surface Area	Basal Metabolic Rate	Respiratory Quotient, Percentage	Weight, Kg.	Estimated Weight, Kg.	Height, M.	Age, Yr.-Mo.
3/ 7/33	3/14-15	1,500	67	15	56	2.4	10.06	8.54	1.21	+0.31	902	40.9	—10	83	21.3	25.4	1.245	9-11
3/25																		
3/26	4/ 5-7	1,650	71	15	62	2.7	11.06	7.53	0.42	+3.11	946	44.3	— 5	82	23.2			
	4/18-20	.....	..	..	..	...	.....	7.42	0.90	+2.74	904	41.8	— 8	81				
5/13	5/ 8-10	.....	..	..	..	...	.....	7.01	0.67	+3.38	936	4.24	— 7	81				
5/14	5/22-24	1,650	67	13	54	2.2	9.61	6.82	1.26	+1.53	953	43.1	— 5	82	24.8	....	.....	10-1
	6/ 1-3	.....	..	..	..	...	.....	6.35	0.63	+2.63								
6/19	6/ 9-11	.....	..	..	..	...	.....	7.15	0.92	+1.54								
6/20	6/23-25	1,650	66	10	41	1.7	7.40	6.04	1.29	+0.07	1,003	45.4	— 2	83	25.0			
	7/ 5-7	.....	..	..	..	...	.....	6.10	1.10	+0.20								
7/16	7/10-12	.....	..	..	..	...	.....	6.38	0.50	+0.52	938	42.5	— 7	84				
7/17	7/22-24	1.800	72	10	45	1.8	8.05	6.18	0.75	+1.12	...	....	....	..	25.0			
	7/26-28	.....	..	..	..	...	.....	6.13	0.55	+1.37	941	42.2	— 7	80				
8/ 4	7/31-8/2	.....	..	..	..	...	.....	6.49	1.55	+0.01								
8/ 5	8/10-12	1,800	70	15	66	2.6	12.07	8.41	0.42	+3.24	...	....	....	..	25.7	....	1.255	
	8/16-18	.....	..	..	..	...	.....	7.66	0.56	+3.85	926	41.1	—10	76				
8/22	8/20-22	.....	..	..	..	...	.....	9.55	0.64	+1.88								
8/23	8/28-30	1,800	71	13	58	2.3	10.51	8.37	0.74	+1.40	...	....	....	..	25.4	....	.....	10-4
	9/ 2-4	.....	..	..	..	...	.....	7.20	0.63	+2.68								
9/ 9	9/ 7-9	... ..	..	..	..	...	.....	7.47	0.88	+2.16								
9/10	9/15-17	2,000	78	13	65	2.6	11.62	7.06	1.19	+3.37	...	....	....	..	25.4			
	9/22-24	.....	..	..	..	...	.....	6.30	0.52	+4.80								
9/29	9/25-28	.....	..	..	..	...	.....	6.19	0.49	+4.94	1,022	45.8	+ 1	80				
9/30	10/ 3-5	2,000	78	15	75	2.9	13.41	5.97	1.03	+6.45	.....	....	....	..	25.4			
	10/ 8-10	.....	..	..	..	...	.....	7.42	0.76	+5.23	1,092	47.8	+ 5	83				
10/18	10/12-14	.....	..	..	..	...	.....	7.62	0.81	+4.98								
10/19	10/29-31	2,000	76	10	50	1.9	8.94	6.60	0.38	+1.96	.....	....	....	..	26.2	....	.....	10-6
	11/ 7-9	.....	..	..	..	...	.....	7.57	0.48	+0.89								
	11/11-13	.....	..	..	..	...	.....	7.80	0.59	+0.54								
12/ 2	11/17-19	.....	..	..	..	...	.....	7.05	0.71	+1.18	1,123	49.1	+ 8	84				
12/ 3	12/ 3-5	2,000*	76	25	125	4.7	20.64†	9.92	1.84	+8.88	.....	....	....	..	26.5	....	1.270	
	12/ 6-8	.....	..	..	..	...	.....	10.47	0.68	+9.49								
12/14	12/10-12	.....	..	..	..	...	.....	10.83	0.93	+8.88	1,099	48.2	+ 6	81				
12/15	12/19-21	2,000*	76	20	100	3.8	17.10†	10.59	0.82	+5.69	.....	....	....	..	26.5			
	12/23-25	.....	..	..	..	...	.....	10.38	0.32	+6.40								
	12/28-30	.....	..	..	..	...	.....	11.77	0.75	+4.58	1,082	47.0	+ 3	85				
	1/11-13	.....	..	..	..	...	.....	13.72	0.84	+2.54								
1/19/34	1/17-19	.....	..	..	..	...	.....	11.23	0.76	+5.01	1,087	46.7	+ 3	84	26.9	28.0	1.280	10-9

* Part of diet refused.
† Represents nitrogen intake after correction for refusals.

2.8 Gm. of protein per kilogram and 81 calories with 2.0 Gm. of protein per kilogram. As in most of our subjects, when 15 per cent of the calories were derived from protein adequate growth was obtained with lower caloric levels than when a lesser percentage of protein was used.

SUBJECT 2.—E. M. (chart 2 and table 2), a white girl, was studied from the age of 9 years and 11 months through the age of 10 years and 9 months.

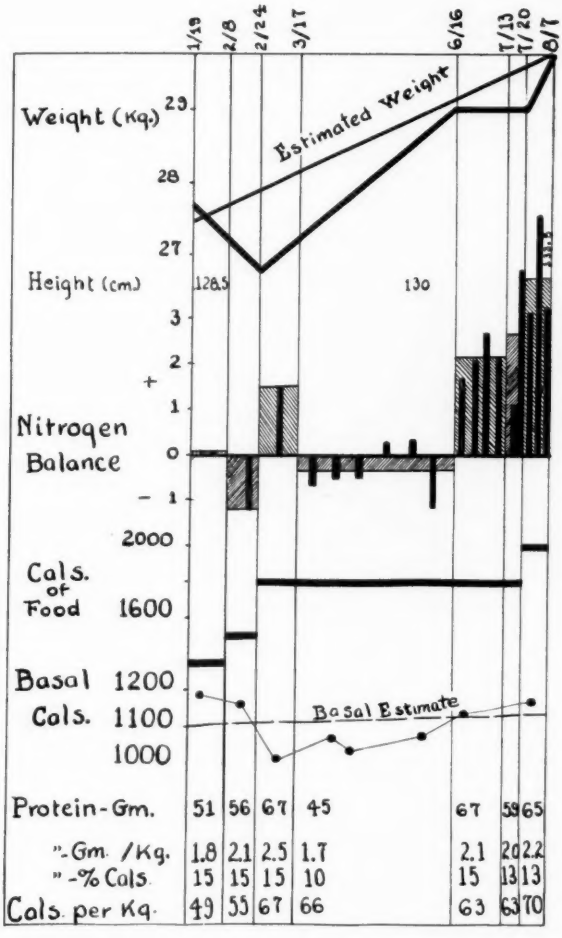

Chart 3.—Results of the study of subject M. S. Note that the subject gained weight for four months, during much of which time she had a negative nitrogen balance.

In the first period, from March 7 to 25, with 1,500 calories there was gain in weight, but the appetite was not satisfied. The calories were increased to 1,650 on March 26 and kept constant until July 16, while the protein was varied from 15 to 13 to 10 per cent of the intake. The nitrogen balance decreased

TABLE 3.—*Observations on M. S. (Female, Born on July 9, 1923)*

Period of Diet	Dates of Three Day Collections	Calories of Food	Calories per Kg. of Weight	Protein, Percentage of Calories	Protein, Gm.	Protein, Gm. per Kg. of Weight	Protein Nitrogen	Nitrogen in Urine	Nitrogen in Stool	Nitrogen Balance	Basal Calories	Basal Calories per Unit of Surface Area	Basal Metabolic Rate	Respiratory Quotient, Percentage	Weight, Kg.	Estimated Weight, Kg.	Height, M.	Age, Yr.-Mo.
1/19/33	1/26-28	1,350	48	15	51	1.8	9.05	7.78	....	+0.12	1,192	50.7	+ 9	76	27.7	27.2	128.5	9-6
2/ 7																		
2/ 8	2/21-22	1,500	55	15	56	2.1	10.06	10.61	....	−1.18	1,161	49.5	+ 6	80	27.2			
2/23																		
2/24	3/ 8-10	1,800	67	15	67	2.5	12.07	8.62	1.94	+1.51	1,015	43.2	− 7	80	26.8			
3/16																		
3/17	3/25-27	1,800	66	10	45	1.7	8.05	7.53	1.19	−0.67	.....	....	....	..	27.2			
	4/14-16	.....	..	..	..	...	.....	7.36	1.15	−0.46	1,073	45.2	− 3	76				
	5/ 6-8	.....	..	..	..	...	.....	7.53	1.03	−0.51	1,034	43.1	− 7	79				
	5/22-24	.....	..	..	..	...	.....	6.57	1.21	+0.27								
	6/ 2-4	.....	..	..	..	...	.....	6.97	0.70	+0.38	1,076	44.8	− 6	77				
6/15	6/11-13	.....	..	..	..	...	.....	8.24	0.96	−1.15	.....	....	....	..	29.0	....	130.0	
6/16	6/24-25	1,800	63	15	67	2.1	12.07	9.82	0.60	+1.65	1,138	47.4	+ 2	77	29.0			
	6/27-29	.....	..	..	..	...	.....	9.03	0.96	+2.08								
	7/ 4-6	.....	..	..	..	...	.....	8.62	0.78	+2.67								
7/12	7/ 9-11	.....	..	..	..	...	.....	8.87	1.06	+2.14								
7/13	7/15-17	1,800	63	13	59	2.0	10.50	8.36	1.04	+1.10	.....	....	....	..	29.0	....	.....	10.0
7/19	7/18-19	.....	..	..	..	...	.....	5.66	0.76	+4.09								
7/20	7/27-29	2,000	70	13	65	2.2	11.62	7.81	0.76	+3.05	1,158	47.3	+ 2	80	29.0			
	8/ 1-3	.....	..	..	.	...	.....	5.03	1.27	+5.32								
8/ 6	8/ 5-7	.....	..	..	..	...	.....	7.60	0.77	+3.87	.....	....	....	..	29.8	....	133.5	

directly with the decrease in protein intake; with 10 per cent of protein gain in weight ceased. With an increase in calories to 1,800 on July 17 and protein still at 10 per cent, there was a rapid spurt in the weight curve. The protein was increased to 15 and 13 per cent, with corresponding changes in the nitrogen balance. After the rapid gain in weight, the child failed to maintain it on a regimen of 1,800 calories. On September 10 the calories were increased to 2,000, with the protein remaining at 13 per cent. Gain in weight did not occur until the protein was increased to 15 per cent. On the 2,000 calory level, protein was varied from 13 to 15 to 10 to 20 to 25 per cent. The child gained adequately with 15 per cent.

This subject, when her intake of protein was increased to 25 per cent, was unable to take all of the diet and suffered from listlessness, loss of appetite and abdominal discomfort. The caloric and protein levels in the periods from March 26 to May 13, from July 17 to August 4 and from October 19 to December 2 were adequate for growth and a positive nitrogen balance in this child. In the first of these periods the basal rate varied from —5 to —8 to —7, in the second it was —7 and in the last it was +8. In a malnourished child gain in weight is not to be taken as a measure of adequate intake of calories, since the lowered metabolic rate permits gain at a level of intake lower than is optimal.

Subject 3.—M. S. (chart 3 and table 3), a white girl, was studied from the age of 9 years and 6 months through the age of 10 years and 1 month.

With an intake of 1,350 and 1,500 calories in the first two periods, respectively, the child lost weight. With an increase to 1,800 calories, she gained weight rapidly. From March 17 to June 15 she gained weight on an intake of which 10 per cent was protein, while the average of the nitrogen balances for the period was negative. From June 16 to July 19, with the calories constant and the protein percentage varying from 15 to 13 per cent, there were a leveling of the weight curve, a positive nitrogen balance and a slight but definite increase in basal calories. With a further increase in calories on July 20, there was a rapid spurt in growth and an increase in the nitrogen balance.

The record for this subject illustrates the possibility of gain in weight with a negative nitrogen balance and a lowered metabolic rate on diets with calories adequate for gain but low in protein. With calories constant and an increase in protein, the metabolic rate rose, there was storage of nitrogen and gain in weight ceased. With an increase in both calories and protein there was gain in weight with a positive nitrogen balance.

Subject 4.—B. B. (chart 4 and table 4), a white girl, was studied from the age of 8 years and 7 months to the age of 9 years.

The child was kept on a constant caloric intake of 1,400 calories, and the percentage of protein was varied from 15 to 13 to 10 to 20 to 25 per cent of the caloric intake. Differing from most of our subjects, she showed a positive nitrogen balance with 10 per cent protein. The case is reported because it shows the interesting phenomenon noted by Talbot and Gamble [9]—the critical point beyond which an increase in intake of protein is followed by diminished storage of nitrogen. The

9. Talbot, F. B., and Gamble, J. L.: Protein Metabolism of an Infant, Am. J. Dis. Child. **12**:333 (Oct.) 1916.

explanation for this may be found in the elevation of the basal metabolic rate which results from the increase in the intake of protein.

The factors, other than the actual number of grams of protein ingested, which affect the nitrogen balance were discussed in the report of the White House Conference. The effect of varying the caloric

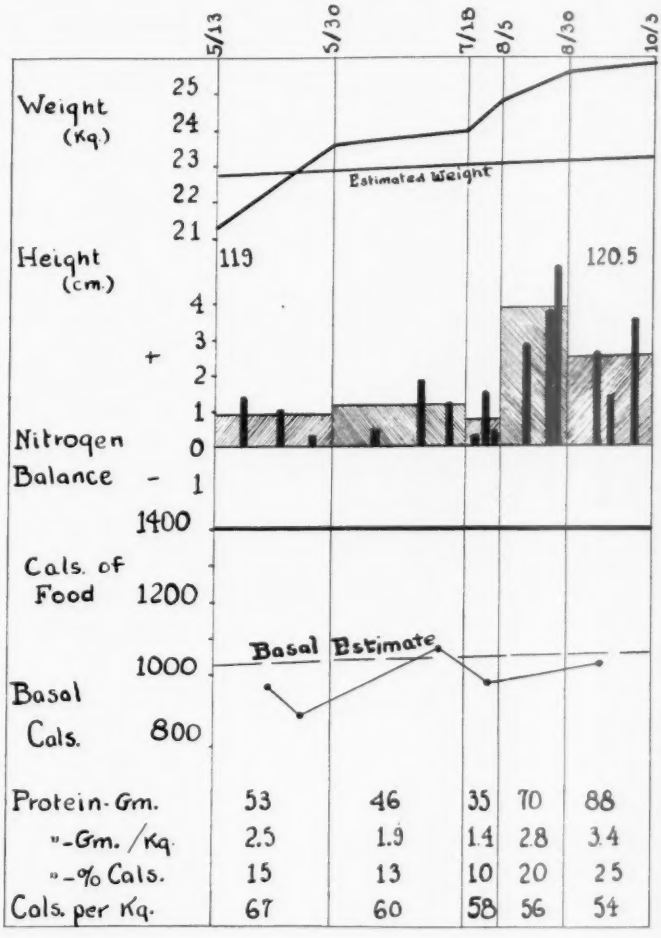

Chart 4.—Results of the study of subject B. B. Note that an increase in the amount of protein ingested beyond 20 per cent is followed by a lessened retention of nitrogen.

intake by increasing the amounts of carbohydrate and of fat was reexamined in this study.

SUBJECT 5.—E. M. (chart 5 and table 5), a white girl (already studied as subject 2), aged 11 years, was maintained on a constant intake of 50 Gm. of

TABLE 4.—*Observations on B. B. (Female, Born on Oct. 6, 1924)*

Period of Diet	Dates of Three Day Collections	Calories of Food	Calories per Kg. of Weight	Protein, Percentage of Calories	Protein, Gm.	Protein, Gm. per Kg. of Weight	Protein Nitrogen	Nitrogen in Urine	Nitrogen in Stool	Nitrogen Balance	Basal Calories	Basal Calories per Unit of Surface Area	Basal Metabolic Rate	Respiratory Quotient, Percentage	Weight, Kg.	Estimated Weight, Kg.	Height, M.	Age, Yr.-Mo.
4/13/33	4/29-5/1	1,400	66.8	15	52.5	2.45	9.39	6.80	1.22	+1.37	965	47.3	— 1	78	21.3	22.7	1.190	8-7
	5/12-14	.....	....	..	....	....	....	7.28	1.04	+1.07	886	42.6	—11	89				
5/29	5/25-27	.....	....	..	....	....	....	7.79	1.28	+0.32								
5/30	6/14-17	1.400	59.8	13	45.5	1.93	8.14	6.96	0.67	+0.51	...	....	....	..	23.6			
	7/ 6-8	.....	....	..	....	....	....	5.40	0.87	+1.87								
7/17	7/15-17	.....	....	..	....	....	....	5.85	1.15	+1.14	1,076	50.9	+ 6	81	....	....	.....	8-9
7/18	7/25-27	1,400	57.6	10	35.0	1.44	6.25	4.55	1.40	—0.30	...	....	....	..	24.0			
	7/29-31	.....	....	.	....	....	....	4.29	0.38	+1.58	974	45.6	— 5	81				
8/ 4	8/ 3-5	.....	....	..	....	....	....	4.80	0.98	—0.47								
8/ 5	8/13-15	1,400	56.0	20	70.0	2.80	12.50	8.73	0.87	+2.90	...	....	....	..	24.8			
	8/19-21	.....	....	..	....	....	.....	7.88	0.89	+3.73								
8/29	8/22-24	.....	....	..	....	....	.....	6.21	1.26	+5:03								
8/30	9/ 8/10	1,400	54.4	25	87.5	3.40	15.65	11.80	1.21	+2.64	...	....	....	..	25.6			
	9/12-14	.....	....	..	....	....	.....	12.54	1.72	—1.39	1,029	47.2	— 2	83				
10/ 3	9/16-18	.....	....	..	....	....	.....	10.73	1.42	+3.53	...	...	....	..	25.8	23.2	1.205	9-0

protein, and the nitrogen balances were noted for three successive twelve day periods with intakes of 1,650, 1,800 and 2,000 calories, the successive increases having been made equally in fat and carbohydrate. With the 1,650 calory level, the nitrogen balance by three day periods became increasingly negative; with 1,800 calories the average of the balances was negative, and with 2,000 calories the balances became increasingly positive.

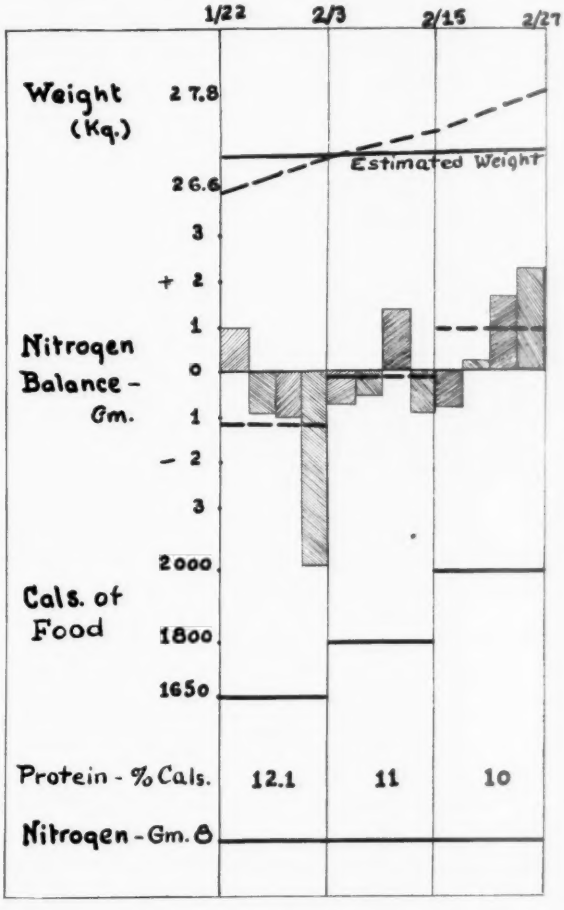

Chart 5.—Results of the study of subject E. M. The shaded areas represent the continuous nitrogen balance charted in three day periods. The broken line represents the average of the balances in each of the twelve day periods. The experiment demonstrates the "sparing effect" on protein of an increase in calories of fat and carbohydrate and illustrates the dependence of the nitrogen balance on the total caloric value of the food mixture.

In the case of this child 1,650 calories were sufficient for gain. With protein constant, however, a definitely positive nitrogen balance did not occur until the calories were increased to 2,000, the percentage of protein in the diet being lower

than that which most of our subjects were found to require. This finding would seem to make desirable the estimation of the protein requirement in terms of percentage of the total calories necessary for gain rather than as a specific number of grams per unit of weight as suggested by Cathcart [10] and Powers.[11]

In tables 6 (girls) and 7 (boys) we have compiled the protein and caloric requirements of 13 girls and 14 boys. The figures were obtained by taking from the chart of each child the lowest caloric and protein level at which there were a positive nitrogen balance and a gain in weight and height approximating the estimated normal growth curve.

TABLE 5.—*Further Observations on E. M. (Female, Born on April 16, 1923)*

	Diet				Nitrogen				
	Calories	Protein	Fat	Carbohydrates	Food	Urine	Stool	Balance	Weight, Kg.
1/22-2/2	1,650	50 Gm. (12.1%)	66	212	8	....	....	.....	26.7
1/23-25	.....	.....	..	...	..	6.57	0.38	+1.05	
1/26-28	.....	.....	..	...	..	8.15	0.67	—0.82	
1/29-31	.....	.....	..	...	..	8.22	0.67	—0.89	
2/ 1-3	.....	.....	..	...	..	11.15	1.05	—4.20	
							Average,	—1.20	
2/3-2/14	1,800	50 Gm. (11.1%)	75	231	8	....	....	.....	27.1
2/ 4-6	.....	.....	..	...	..	7.89	0.74	—0.63	
2/ 7-9	.....	.....	..	...	..	7.53	0.96	—0.49	
2/10-12	.....	.....	..	...	..	5.49	0.98	+1.53	
2/13-15	.....	.....	..	...	..	7.98	0.82	—0.80	
							Average,	—0.10	
2/15-26	2,000	50 Gm. (10%)	86	256	8	....	....	.....	27.4
2/16-18	.....	.....	..	...	..	7.75	0.97	—0.72	
2/19-21	.....	.....	..	...	..	6.49	1.14	+0.37	
2/22-24	.....	.....	..	...	..	5.36	0.98	+1.66	
2/25-27	.....	.....	..	...	..	5.00	0.70	+2.30	
							Average,	+0.90	27.8

The basal metabolic rates at each level were recorded. In some instances in which the interval of time was long we have shown the apparent requirements of the same child at different ages or at different weights or basal metabolic levels; in some cases the requirements of growth and storage of nitrogen were met at varying levels depending on the interrelationship of protein and calories.

10. Cathcart, E. P.: Influence of Fat and Carbohydrate on the Nitrogen Distribution in the Urine, Biochem. J. **16**:247, 1922.

11. Powers, G. F.: Comparison and Interpretation on Caloric Basis of Milk Mixtures Used in Infant Feeding, Am. J. Dis. Child. **30**:453 (Oct.) 1925.

TABLE 6.—*Observations on Girls*

Subject	Age, Yr.-Mo.	Height, In.	Calories of Food			Protein		Basal Metabolic Rate	Basal Calories		Increment, Percentage of Calories Above Basal	Weight	
			Per In. of Height	Per Kg. of Weight	Total Number	Gm. per Kg. of Weight	Percentage of Calories		Per Sq. M. per Hr.	Total for 24 Hr.		Actual, Lb.	Estimated, Lb.
J. C.	5- 6	43.0	34.9	69	1,500	3.3	13	+ 2	52.7	1,000	50	48	41
E. H.	6-11	46.8	35.1	60	1,650	3.2	13	....	....				
	7- 8	49.3	33.5	..	1,650	3.2	15	—13	43.2	1,004	65	60	56
M. M.	8- 0	49.5	33.3	69	1,650	2.2	13	— 1	47.3	1,063	55	53	54
	8- 6	51.0	39.2	70	2,000	2.6	15	+ 1	48.5	1,162	72	63	62
J. McK.	8- 4	50.5	34.7	60	1,750	2.1	14	— 2	46.8	1,116	57	65	59
G. M.	8- 5	50.0	33.0	55	1,650	1.8	13	— 7	44.7	1,082	53	65	55
M. H.	8- 5	50.5	39.6	63	2,000	2.4	15	—12	42.1	1,061	88	70	60
B. B.	8-10	47.0	30.0	58	1,400	1.4	10	— 5	45.6	974	43	53	51
A. A.	9- 5	49.3	33.0	60	1,650	1.9	13	+ 1	46.9	1,093	51	61	55
	9-11	49.8	36.0	59	1,800	1.9	13	+ 4	48.5	1,176	53	68	59
M. S.	9- 8	51.0	35.0	67	1,800	2.5	15	— 7	43.2	1,015	77	59	60
	10- 6	54.0	37.0	63	2,000	1.9	13	+10	50.1	1,336	50	75	71
E. M.	10- 0	49.0	33.6	71	1,650	2.7	15	— 5	44.3	946	74	51	56
	10- 3	49.5	36.0	72	1,800	1.8	10	— 7	42.2	941	91	55	58
	10- 6	50.0	40.0	76	2,000	1.9	10	+ 8	49.1	1,123	78	58	59
N. C.	10- 4	55.3	43.0	70	2,400	2.6	15	— 7	42.0	1,298	85	77	75
A. C.	11- 2	54.0	37.0	61	2,000	1.5	10	—13	39.9	1,049	91	72	66
	11- 4	54.7	36.5	58	2,000	2 2	15	— 7	41.3	1,150	74	76	70
J. L.	12- 5	60.7	36 2	55	2,200	2.1	15	— 5	41.0	1,292	70	90	100

TABLE 7.—*Observations on Boys*

Subject	Age, Yr.-Mo.	Height, In.	Calories of Food			Protein		Basal Metabolic Rate	Basal Calories		Increment, Percentage of Calories Above Basal	Weight	
			Per In. of Height	Per Kg. of Weight	Total Number	Gm. per Kg. of Weight	Percentage of Calories		Per Sq. M. per Hr.	Total for 24 Hr.		Actual, Lb.	Estimated, Lb.
C. B.	4- 5	41.0	34.2	82	1,400	3.1	15	....	....	.....	..	38	38
R. R.	5- 5	41.0	34.1	77	1,400	2.5	13	— 6	49.5	806	74	38	38
R. B.	5- 9	48.0	34.3	71	1,650	1.8	10	—15	44.7	965	70	52	52
	6- 0	48.3	34.1	70	1,650	2.3	13	— 7	49.5	1,068	55	53	53
M. G.	5-10	40.5	30.9	74	1,250	2.8	15	+ 1	53.7	876	43	37	37
	6- 1	41.25	40.0	90	1,650	2.2	10	+ 6	56.2	972	70	41	38
	6- 6	42.5	38.8	81	1,650	2.1	10	— 3	51.6	936	76	44	39
J. McL.	6- 0	48.0	41.2	85	2,000	2.4	10	—10	47.0	1,015	97	52	52
	6- 1	48.3	41.2	83	2,000	2.7	13	± 0	52.2	1,126	78	53	52
R. G.	7- 0	49.0	33.7	66	1,650	2.5	15	— 4	51.1	1,128	46	56	55
E. K.	9- 7	52.0	38.4	71	2,000	2.7	15	— 2	49.2	1,150	74	62	64
	9- 9	52.5	45.7	82	2,400	2.6	13	— 1	49.3	1,218	97	64	66
	9-11	52.5	41.2	71	2,200	2.7	15	— 3	48.4	1,243	77	68	66
J. P.	9- 8	53.5	34.0	67	1,800	2.2	13	—10	44.9	1,097	64	59	68
	10- 1	54.5	40.0	68	2,200	2.7	15	— 7	45.6	1,226	79	71	71
D. D.	9-10	52.5	38.0	71	2,000	2.7	16	— 1	48.6	1,188	68	62	64
	10- 0	53.0	45.0	80	2,400	2.0	10	— 4	47.2	1,212	98	69	67
G. F.	11- 5	56.0	42.8	64	2,400	1.6	15	— 9	44.3	1,330	80	83	77
	11- 7	56.5	38.9	53	2,200	2.6	20	— 5	45.9	1,411	56	92	79
D. C.	11- 3	58.0	48.3	58	2,800	2.3	15	+ 6	50.5	1,576	78	90	84
J. C.	11- 9	55.3	43.3	83	2,400	3.1	15	+ 2	48.5	1,271	89	64	74
N. S.	12- 5	56.5	42.4	76	2,400	2.9	15	+ 3	48.9	1,340	79	70	80
J. McC.	13- 6	64.5	50.0	50	3,200	1.4	11	— 5	44.5	1,816	76	144	116

COMMENT

Our subjects showed marked individual variation in the apparent caloric requirement. Graphs of calories per kilogram of weight, calories per inch of height and protein per kilogram of weight failed to approach smooth curves. As was indicated in the report of the White House Conference, calories per inch of height more nearly approached a constant than the other data. In most individual cases the apparent requirement in terms of calories per kilogram fell between the standard of Holt and Fales [12] and that of Camerer [13] and simulated closely that of Gillett.[14] The increment for activity over basal calories, averaging 74 per cent for the boys and 67 per cent for the girls, was higher than that observed by Camerer and others and lower than that noted by Müller.[15]

Our particular interest has been the protein requirement. The question as to what storage of nitrogen is optimal is not answerable. In terms of the amount theoretically required for the synthesis of a given quantity of tissue, the retentions here recorded are high. It was possible, however, clearly to define minimal and maximal quantities, the former as the point below which the balance was negative, the latter usually as a point beyond which there appeared definite evidences of intoxication. Occasionally there was noted the point mentioned by Gamble and Talbot —a diminished retention with increased intake—but usually before this was reached symptoms of intolerance appeared. The apparent protein requirement per kilogram approached closely that reported by Holt and Fales. With calories sufficient for gain in weight, amounts of protein representing 15 per cent of the total calories seemed adequate. Lesser amounts too frequently resulted in negative balances, in some instances in the face of gain in weight. Amounts larger than 15 per cent did not appear to be necessary; in some subjects such amounts failed to produce increased retention of nitrogen, and in others they produced apathy, pallor, anorexia and abdominal discomfort. Because of the interrelation of calories to protein it seems to us most satisfactory to state the protein requirement in terms of the percentage of calories allotted to protein that is necessary for normal metabolism and growth.

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12. Holt, L. E., and Fales, H. L.: Food Requirements of Children: Total Caloric Requirements, Am. J. Dis. Child. **21**:1 (Jan.) 1921.

13. Camerer, W., cited by Holt and Fales.[8]

14. Gillett, L. H.: Survey of Evidence Regarding Food Allowances for Healthy Children, New York, New York Association for Improving the Condition of the Poor, 1917.

15. Müller, E.: Metabolism in Children, in von Pfaundler, M., and Schlossmann, A.: Diseases of Children, translated by M. G. Peterman, Philadelphia, J. B. Lippincott Company, 1935, vol. 1, p. 338.

RESULTS

The dietary intake of 13 girls, aged from 5 to 12 years, and 14 boys, aged from 4 to 14 years, was measured over periods averaging nine and four-tenths months; their nitrogen balances were noted for an average of one hundred and eleven days for each child, and periodical observations on their basal metabolism were made. Caloric and protein requirements per unit of height and weight were recorded for each child.

In our subjects gain in weight and height was obtained with caloric intakes representing increments over the basal calories averaging 67 per cent for the girls and 74 per cent for the boys.

With calories adequate for growth, intakes of protein representing 15 per cent of the total calories seemed optimal. Amounts greater than 20 per cent were usually productive of pallor, loss of appetite, abdominal discomfort and sometimes vomiting. With amounts less than 15 per cent a negative nitrogen balance or depression of the basal metabolism was frequently observed.

The so-called sparing effect of fat and carbohydrate on protein was reexamined and confirmed; this constitutes an argument for recording the protein requirement in terms of the percentage of calories rather than of grams per unit of weight.

Dr. O. H. Gaebler analyzed the diets; Dr. F. W. Hartman supervised the determinations, and Miss Ruth Smithyman, Miss Evelyn Foster and Miss Hildegard Heinicke made the calculations of the diets.

TUBERCLE BACILLI IN THE GASTRIC WASHINGS OF INFANTS AND OF CHILDREN

DEMONSTRATION WHEN THE CUTANEOUS REACTION TO TUBERCULIN IS POSITIVE BUT THERE IS NO EVIDENCE OF TUBERCULOSIS ON PHYSICAL OR ROENTGENOGRAPHIC EXAMINATION

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That pulmonary tuberculosis may occur in infants and in children with few if any local or constitutional manifestations has been universally recognized. The diagnosis must necessarily rest on the demonstration of tubercle bacilli in the sputum. It is difficult and in most cases impossible to obtain sputum from infants and from younger children, since such young patients do not expectorate but swallow the sputum and the coughed-up mucus. Examination of the gastric washings for tubercle bacilli, therefore, has become a diagnostic measure of importance in cases of suspected pulmonary tuberculosis in infants and in children. The procedure is a simple one and can be carried out readily.

Gastric lavage with examination of the sediment obtained by centrifuging the stomach washings in cases of tuberculosis was first described by Meunier,[1] in 1898. Meunier's method was not employed extensively until interest in the procedure was reawakened by the publication of Armand-Delille and Vibert's [2] report in 1927. These observers homogenized the sediment with sodium hydroxide before staining it with Ziehl-Neelsen's stain. By this modification they were able to demonstrate tubercle bacilli in the gastric washings in 31 per cent of a series of 110 cases, whereas tubercle bacilli were demonstrated in the gastric washings in only 10.7 per cent of a series of 62 cases in which homogenization of the sediment had not been done. Subsequently, with a series of 500 cases, Armand-Delille [3] was able to demon-

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1. Meunier, H.: Bacilloscopie des crachats extraits de l'estomac pour le diagnostic de la tuberculose pulmonaire, Presse méd. **6**:81-82 (Aug. 13) 1898.

2. Armand-Delille, P. F., and Vibert, J.: Le diagnostic bactériologique de la tuberculose pulmonaire des jeunes enfants par l'examen du contenu gastrique, Presse méd. **35**:402-404 (March 15) 1927.

3. Armand-Delille, P. F.: L'importance de la recherche du bacille de Koch dans le contenu gastrique pour le diagnostique de la tuberculose pulmonaire chez les jeunes enfants, Paris méd. **1**:18-20 (Jan. 3) 1931.

strate tubercle bacilli in the gastric contents of 100 per cent of the patients who showed both auscultatory and roentgenographic signs of caseous processes in the lungs and in the gastric contents of 60 per cent of the patients for whom roentgen examinations furnished the only evidence of tuberculosis.

In 1929, Poulsen, Jensen and Husted [4] supplemented Armand-Delille's modification of Meunier's method with cultivation of the sediment on substrates (using Petroff's medium) and with inoculation of the sediment into guinea-pigs. Other observers (Baumann,[5] Opitz,[6] Jachnis and Zilber,[7] Wallgren [8] and Kereszturi and her co-workers [9]) combined the microscopic examination of the sediment with inoculation into guinea-pigs.

Numerous reports on this subject have since appeared in the literature. While the results on the whole are surprisingly good, the actual percentages of positive findings vary considerably. The variations may be more readily understood if several factors are taken into consideration in interpreting the results obtained. In the first place, the number of instances in which tubercle bacilli are demonstrated by examination of the gastric contents must necessarily vary with the type of patient examined. If the report is based on examinations conducted in a hospital or sanatorium devoted primarily to the care of patients who are frankly tuberculous or who have had definite contact with and recent exposure to persons with active tuberculosis, a higher percentage of positive results may be expected than if it is based on data on the same number of patients in similar age groups who have been examined according to routine on admission to a general hospital, clinic or home not specifically devoted to the treatment of tuberculosis. In the second place, the incidence of positive findings on gastric lavage will vary with the actual prevalence of tuberculosis in the immediate community from which the patients are drawn. It is well known that tuberculosis has

4. Poulsen, V.; Jensen, K. A., and Husted, E.: The Demonstration of Tubercle Bacilli in Small Children with Pulmonary Tuberculosis, Am. J. Dis. Child. **37**:900-908 (May) 1929.

5. Baumann, T.: Die Diagnostik der offenen Lungentuberkulose im Säuglings- und Kindesalter, Beitr. z. Klin. d. Tuberk. **79**:717-723, 1932.

6. Opitz, H.: Neue Forschungen über die Diagnose der Lungentuberkulose durch Bazillennachweis, Acta pædiat. **11**:186-187, 1930.

7. Jachnis, B., and Zilber, S.: Zur Diagnostik der offenen Formen der Lungentuberkulose in frühen Kindesalter, Vrach. delo **13**:269-271, 1930; abstr., Zentralbl. f. d. ges. Kinderh. **25**:320, 1931.

8. Wallgren, A.: Tubercle Bacilli in Children with Erythema Nodosum, Am. J. Dis. Child. **41**:816-822 (April) 1931.

9. Kereszturi, C.; Hauptmann, D.; Schick, B., and Mishulow, L.: Acid-Fast Bacilli in the Stomach Lavage and Feces of Tuberculous Children, J. A. M. A. **100**:1481-1484 (May 13) 1933.

been more prevalent in certain countries as a result of poor economic conditions and as an aftermath of the malnutrition in those districts during the World War. Any section of the population living under poor conditions will show a higher incidence of tuberculosis than a similar group in another locality living under better conditions; and, correspondingly, if the hospital's clientele is drawn from the former group, a higher incidence of tubercle bacilli in the gastric contents will probably be noted. In the third place, the number of gastric analyses done on each patient is an important factor. Poulsen and Andersen [10] have shown that a second and third lavage will raise the number of positive results obtained. In a series of 199 cases in which they were able to demonstrate tubercle bacilli, the bacilli were found on the first lavage in 77 per cent of the cases. A second lavage raised the percentage of positive findings to 91 per cent; a third lavage, to 96 per cent; a fourth lavage, to 98 per cent, and a fifth lavage, to 100 per cent. For practical purposes these investigators have advised examination, in most cases, of the sediment obtained by three separate lavages before the patient is pronounced free from tubercle bacilli. The fact that a difference of almost 20 per cent was obtained by increasing the number of lavages from one to three illustrates the importance of the number of lavages done in each case in interpreting the results obtained by the various observers. Many reports are based on a series of cases in which a single lavage was done or on a series in which more than one lavage was done in only a few cases. On the other hand, in other series reported, several lavages were done in each case.

A fourth factor is the age of the children examined. It is commonly recognized that a positive cutaneous reaction to tuberculin is of more significance the younger the patient, since the tuberculous infection is more recent in origin. Similarly, the younger the group of patients with positive cutaneous reactions, the more likelihood there is of finding tubercle bacilli in the gastric contents, for the infection has probably been of shorter duration and there is less possibility that the initial focus has healed. Table 1 (adapted from the figures given by Poulsen and Andersen [10]) illustrates the influence of age as a factor in the determination of the positive findings of tubercle bacilli in the gastric contents of children with positive cutaneous reactions to tuberculin. It may be seen that bacilli were demonstrated in six sevenths of the cases in which the patient was in the first year of life (the youngest patient being between 1 and 2 months of age); in almost half of the

10. Poulsen, V., and Andersen, A. O.: Four Year's Experience with Examination of Material Obtained by Gastric Lavage: Demonstration of Tubercle Bacilli and Its Significance in Prognosis, Therapy and Estimation of Danger of Infection, Am. J. Dis. Child. **47**:307-321 (Feb.) 1934.

cases in which the patient was in the second year of life, and in one third of those in which the patient was in the third year of life. From the fourth year of life on, the incidence varied from one-tenth for the patients in the ninth year of life to one-fourth for those in the sixth year of life; the average incidence was approximately one-sixth for the patients in each year of life between the third and the eleventh.

Therefore, it may be concluded that the type of patient examined (whether actively tuberculous, with frank signs, or whether recently exposed to active tuberculosis), the type of locality from which the particular hospital draws its patients, the number of lavages employed before the patient is pronounced free from tubercle bacilli and the age of the patients examined are all factors of great practical importance in determining the incidence of tubercle bacilli in the gastric contents in any series of cases reported in the literature.

TABLE 1.—*Distribution of Patients with Tubercle Bacilli in Gastric Contents According to Age (Adapted from Poulsen and Andersen)*

Year of Life	Number of Patients Examined	Tubercle Bacilli in Gastric Contents	Percentage of Positive Findings
1st	62	54	84.0
2d	108	50	46.0
3d	119	38	32.0
4th	94	18	19.0
5th	56	7	12.5
6th	44	11	25.0
7th	31	5	16.0
8th	45	8	17.7
9th	30	3	10.0
10th	21	3	14.0
11th	12	2	16.6
Total	622	199	32.0

A further consideration now arises. What is the significance of one's finding tubercle bacilli in the gastric washings? To rule out the possibility that the bacilli were present as chance invaders in the fauces or in the stomachs of children, just as the diphtheria bacillus, the meningococcus or the pneumococcus may be present in the throats of healthy children, examinations were made of the gastric washings of a control series of children whose cutaneous reactions to tuberculin were negative. Poulsen and Andersen [10] examined the gastric washings of 158 patients with negative cutaneous reactions to tuberculin and were unable to demonstrate tubercle bacilli in any of the washings, although bacilli were found in the washings of 199 (32 per cent) of the 622 children in whom a positive cutaneous reaction to tuberculin had been observed. Similarly, Levin [11] reported on a control series of 40 patients

11. Levin, N.: Étude clinico-bactériologique sur la possibilité de déceler la présence de bacilles tuberculeux chez l'enfant par la méthode du lavage de l'estomac, Svenska läk.-sällsk. handl. **60**:1-108, 1934.

with negative cutaneous reactions to tuberculin, for none of whom tubercle bacilli were found in the gastric washings, whereas bacilli were demonstrated in the gastric contents of 159 (43.5 per cent) of the 365 patients with positive cutaneous reactions to tuberculin of whom roentgenographic examination had been made.

It may therefore be concluded that tubercle bacilli are not chance invaders of the gastric contents. Their presence must mean that there is an active focus from which they are being discharged into the stomach. If primary tuberculosis of the tonsils and adenoids, as well as tuberculosis of the stomach, can be ruled out (by physical and histologic examination after tonsillectomy for the former and by the rarity of the occurrence of the latter), it is evident that the source of the tubercle bacilli in the gastric contents must be an active pulmonary lesion or a tuberculous lymph node which has ulcerated through a bronchus. After coughing, infants and young children, who do not expectorate, swallow the sputum containing the tubercle bacilli, with resultant contamination of the gastric contents.

The pediatric service at the Lenox Hill Hospital admits infants and children for the diagnosis and treatment of all types of childhood diseases except the exanthems. It has been the custom to determine as a matter of routine the reactivity of the skin to tuberculin (by the Pirquet test and if that is negative by Mantoux tests with tuberculin of varying strength). Whenever a positive cutaneous reaction to tuberculin has been obtained, a roentgenogram of the chest has been taken. For almost all of the patients in whom no clinical evidence of pulmonary tuberculosis was found on physical examination, the roentgenogram has been reported as "negative" or as showing intensification of either the lung root shadows or the hilar markings. The problem would then arise as to whether an infant or child with a positive cutaneous reaction to tuberculin in the absence of positive clinical and roentgenographic evidence of tuberculosis should be considered a potential danger to other children with negative cutaneous reactions to tuberculin. Formerly, with infants and young children, who could not expectorate, the pharynx was titillated with a swab so that the patient had a cough reflex, after which sputum was obtained; the sputum was then examined by smear for tubercle bacilli. Unfortunately, a negative result was inconclusive and did not rule out the presence of an active tuberculous lesion. About six years ago, a study was begun at this hospital to determine what proportion of the children with positive cutaneous reactions to tuberculin but with negative physical and roentgenographic findings (and therefore clinically not suspected of having tuberculosis) actually harbored tubercle bacilli in the gastric contents.

The material in this study consisted of all infants and children admitted to the pediatric wards of the Lenox Hill Hospital in whom cutaneous reactions to routine tuberculin tests were found to be positive and in whom no evidence of tuberculosis could be demonstrated on physical and roentgenographic examinations. Patients with roentgenographic evidence of pulmonary tuberculosis and patients with tuberculosis of the bones, joints, glands or skin for whom examinations of the gastric contents for tubercle bacilli were done, were not included. When a child was found to have an infection of the upper part of the respiratory tract or pneumonia, the lavage was not done till the child had been afebrile for several days, so that the death of the guinea-pig of an intercurrent infection caused by the streptococcus or the pneumococcus might be avoided. A single lavage was done in all instances save a few in which it was feasible to do two or three lavages. Although the necessity for multiple examinations of the gastric contents in cases in which tubercle bacilli were not demonstrated on single lavage was recognized, unfortunately, in most instances, by the time autopsy was performed on the guinea-pig (six weeks after the inoculation) the patient had been discharged from the hospital. Toward the end of the study, this problem was finally solved by washing out the stomach on two successive mornings and pooling the two specimens for examination.

The gastric lavage was done while the patient was fasting. The stomach was washed with 200 cc. of physiologic solution of sodium chloride. The washings were then centrifugated, and a smear of the sediment was examined with the Ziehl-Neelsen stain. The remainder of the sediment was suspended in 2 cc. of sterile saline solution, which was then injected into a guinea-pig (intraperitoneally in most instances and subcutaneously in the inguinal region in the remainder). Six weeks later the guinea-pig was killed and examined for evidence of tuberculosis. At necropsy, typical generalized tuberculosis was found in some of the cases in which the results were positive. However, in others only slight enlargement of the regional lymph glands was observed on gross examination (of the inguinal lymph nodes when the subcutaneous route had been employed and of the peritoneal lymph nodes when an intraperitoneal inoculation had been given); on microscopic examination of the lymph nodes, tubercle bacilli were seen. The spleen was usually enlarged when evidence of tuberculosis was present. On gross examination of the liver and spleen, whitish, tubercle-like lesions were observed on section; on microscopic examination, tubercles were demonstrated.

The sediment obtained from the gastric washings was inoculated into guinea-pigs in 86 cases in which an infant or child was found to have a positive cutaneous reaction to tuberculin but in which no clinical evidence of tuberculosis could be demonstrated on either physical or roentgenographic examination. In 7 of the cases, tubercle bacilli were present in the gastric contents, as was demonstrated six weeks later, at necropsy, by the observation of tuberculosis in the inoculated animal. In 3 cases, examination of the guinea-pig at necropsy revealed suggestive, tubercle-like lesions, but an absolute histologic diagnosis of tuberculosis could not be made. In 76 cases, no evidence of tuberculosis was found in the inoculated animal.

In 10 additional cases, forming a small control group, in which the cutaneous reactions to tuberculin (Pirquet and Mantoux tests with doses up to 1 mg. in strength) were negative and in which no evidence of tuberculosis was found on physical and roentgen examination, the sediment obtained from the gastric washings was injected into a guinea-pig. In none of the cases in which the cutaneous reaction to tuberculin was negative was tuberculosis produced in the inoculated animal.

Thus, in approximately one twelfth of the cases (7 of the 86 cases) in which the cutaneous reaction to tuberculin was positive but in which there was no physical or roentgenographic evidence of tuberculosis, examination of the sediment of the gastric washings by inoculation into a guinea-pig revealed the presence of tubercle bacilli in the original gastric contents. This indicates the presence in 8.14 per cent of the cases comprising the series of an active tuberculous focus either in the lungs or in the hilar or tracheobronchial lymph nodes, which would otherwise have been unrecognized.

Since tubercle bacilli were present in the gastric washings of these patients, the bacilli could have been transmitted to other persons by coughing or vomiting. Such patients are therefore an actual source of danger to children who have been shown to be free from tuberculous infection by negative cutaneous reactions to tuberculin. They must be segregated and completely isolated to prevent infection of an uninfected child.

COMMENT

That a slightly higher percentage of positive results might have been obtained by multiple lavage examinations is likely, in view of Poulsen and Andersen's demonstration that three lavages increased the efficacy of the test from 77 per cent on the first lavage to 96 per cent on the third lavage. However, it must be remembered that the children in this series were not clinically or roentgenographically tuberculous. In fact, had not routine cutaneous tests with tuberculin been part of the regimen of the service, most of these children would not even have been suspected of being infected. Furthermore, even with the positive cutaneous reaction to tuberculin, the diagnosis of an active tuberculous focus could not have been made in any of the cases without the evidence obtained by examination of the gastric contents. In Poulsen and Andersen's group of 199 cases in which results were positive, auscultatory and roentgenographic evidences of tuberculosis were found in 108 cases and roentgenographic evidence alone in 46 cases. In 14 cases no roentgenogram was taken, and only in the remaining 31 cases was there no evidence of tuberculosis either in the roentgenogram or on physical examination. The group of 31 cases, representing but 15.6 per cent of Poulsen and Andersen's cases in which results were positive, comprises the only part of their series which can be compared with our series, since these 31 cases are the only instances of the entire series in which the diagnosis of pulmonary tuberculosis could be made solely on the basis of the demonstration of tubercle bacilli in the gastric contents. Unfortunately, no mention was made in the report by Poulsen and Andersen of the total number of children examined for whom physical and roentgen findings were negative and cutaneous reactions to tuberculin positive, of which these 31 must form only a part. Consequently, no direct percentage comparison can be made with our figures.

Friedman, Esserman and Black [12] published a somewhat similar study in 1933, based on the examination of the gastric washings of 60 children in whom no physical signs of tuberculosis could be found. According to their statement, 26.7 per cent of the patients (16 cases) had negative cutaneous reactions to tuberculin, and 73.3 per cent (44 cases) had positive reactions. On roentgenographic examination, only 45 children had either questionable findings or no evidence of tuberculosis, and of this group 14 patients had negative cutaneous reactions to tuberculin. Consequently, it may be concluded that in 31 of their cases in which the cutaneous reaction to tuberculin was positive, the physical and roentgenographic examinations gave essentially negative results. These 31 cases, therefore, form a group which can be compared with our series. In only 2 of the entire 60 cases were tubercle bacilli demonstrated in the gastric washings; both occurred in boys in whom the cutaneous reaction to tuberculin was positive and for whom roentgenograms showed questionable involvement of the tracheobronchial lymph nodes. These 2 cases, in which the diagnosis could only have been made by the examination of the gastric contents, belong to the group of 31 cases previously mentioned and therefore represent an incidence of 6.45 per cent, which may be compared with the 8.14 per cent of similar results in the series reported here.

Armand-Delille [13] mentioned the finding of tubercle bacilli in the gastric contents of 2 patients who had been in contact with tuberculous parents and in whose roentgenograms only small hilar shadows were found. Unfortunately, he did not state any other figures on this point. Kereszturi and her co-workers, however, were unable to demonstrate tubercle bacilli in the gastric washings of 25 patients with positive cutaneous reactions to tuberculin but without any evidence of tuberculosis on physical and roentgenographic examination.

It is of interest to analyze further the cases reported here. Of the 7 patients for whom gastric examination gave positive results, all but 2 were boys. Their ages were 1½, 1¾, 2, 2½, 3, 3½ and 9 years, respectively. There was a history of familial incidence of tuberculosis in only 2 of the 6 cases. Actual direct contact with and exposure to tuberculosis in the family or in outsiders was known to have occurred in the same 2 instances. Of the children whose gastric washings showed nothing abnormal, 35 were girls and 41 were boys. Their ages ranged

12. Friedman, E.; Esserman, A. L., and Black, M. H.: Tubercle Bacilli in the Sputum of Children Free from Manifest Pulmonary Tuberculosis (as Determined by Examination of Gastric Sediment), J. Pediat. **2**:283-287, 1933.

13. Armand-Delille, P. F.: Pulmonary Tuberculosis in Infants: Bacteriologic Diagnosis by Examination of the Stomach Contents, Am. J. Dis. Child. **34**:547-548 (Oct.) 1927.

from less than 1 year to 15 years. A history of familial incidence of tuberculosis was given in 28 of the cases, and actual direct contact with or exposure to tuberculosis had occurred in 25. Malnutrition was a finding in 4 of the 7 cases in which the results were positive, in all 3 of the cases in which they were questionably positive and in 57 of the 76 cases in which they were negative.

Of the 10 patients with negative cutaneous reactions to tuberculin, comprising the small control series, 5 had malnutrition. A family history of tuberculosis was elicited in 2 instances. Actual direct exposure to tuberculosis was known to have occurred in 1 case.

Roentgenographic examination did not reveal evidence of active tuberculosis in any of the patients with a positive cutaneous reaction to tuberculin. In 1 instance in each of the groups with positive or with negative results with guinea-pigs, a slight parenchymal infiltration was observed. A Ghon focus was seen in one of the cases in which results were questionably positive and in 7 of the cases in which tests with guinea-pigs were negative but in none of the cases in which the test of the gastric washings was definitely positive. In no case in the entire group of 86 cases in which the cutaneous reaction to tuberculin was positive was the roentgenologist willing to affirm the presence of active tuberculosis, although he had been informed in each instance of the positive cutaneous reaction to tuberculin. In the majority of the cases (65 cases), the reported hilar thickening or intensification of linear markings was no more than that often seen in roentgenograms of the chest of nontuberculous children, particularly those with a mild sinus infection or those who have had repeated attacks of infection of the upper respiratory tract or of bronchitis. It is of interest that for the control group of 10 uninfected children with negative cutaneous reactions to 1 mg. of old tuberculin, the roentgenograms were reported as showing similar intensification of linear markings and heaviness of hilar markings. The data of interest has been arranged in table form so that the main points of significance may be more readily observed (tables 2 and 3).

Brief consideration must be given to the several important advantages of examination of the gastric washings for tubercle bacilli in children. The results obtained are of diagnostic, prognostic and therapeutic significance. The method is useful and suitable for the demonstration of tubercle bacilli in infants and children at an age when expectoration does not occur and when it is difficult and in most cases impossible to obtain any sputum for examination. With this method of examination, a diagnosis of pulmonary tuberculosis may be made even if the condition is in the preallergic stage (prior to the development of a positive cutaneous reaction to old tuberculin). This occurred

TABLE 2.—*Demonstration of Tubercle Bacilli in Gastric Washings of In[illegible] and Chil[illegible]*

Case No.	Name of Patient	Age, Years	Sex	Malnutrition	Symptoms Leading to Tuberculin Tests	Cutaneous Reaction to Tuberculin		Roentgenographic Findings						Family History of Tuberculosis	Pa[illegible]
						Pirquet	Mantoux	Heavy Lung Root Shadows	Exaggeration of Hilar Markings	Hilar Adenopathy	Calcification in Lung Roots	Ghon Focus	Parenchymal Infiltration		
1	A. S.	2	♂	++	Low grade fever; malnutrition	+	..	+	0	+	0	0	0	0	
2	G. P.	9	♂	+	Recurrent colds for 1 year; malnutrition	+	..	++	0	0	0	0	+ (Slight in left hilus)	0	
3	C. A.	3	♂	+	Frequent infections of upper part of respiratory tract; malnutrition	+	..	+	0	0	+	0	0	0	
4	A. L.	3½	♂	0	Listlessness; history of exposure to tuberculosis	+	..	+	+	0	+	0	0	(Maternal aunt) +	
5	D. D.	2½	♂	0	Frequent colds; chronic infection of tonsils	+	..	+	+	++	0	0	0	(Maternal aunt) +	
6	M. R.	1½	♀	0	Small, olive-size preauricular node present for 3 mos.	Negative	+ (0.5 mg., negative; 1 mg., positive)	+	+	0	0	0	0	0	
7	J. C.	6	♂	+	Malnutrition	+	..	+	+	0	+	0	0	0	

TABLE 3.—*Cases in Which There Was no Clinical or Roentgenographic Evidence of Tuberculosis [illegible] in Which*

Case No.	Name of Patient	Age, Years	Sex	Malnutrition	Symptoms Leading to Tuberculin Tests	Cutaneous Reaction to Tuberculin		Roentgenographic Findings						Family History of Tuberculosis	Pa[illegible]
						Pirquet	Mantoux	Heavy Lung Root Shadows	Exaggeration of Hilar Markings	Hilar Adenopathy	Calcification in Lung Roots	Ghon Focus	Parenchymal Infiltration		
1	W. N.	6½	♂	+	Malnutrition	+	..	+	+	0	+	+	0	(Father) +	
2	P. A.	6½	♂	+	Malnutrition	+	..	+	+	+	+	0	0	+	
3	M. N.	5	♂	+	Malnutrition	Negative	+	+	0	0	+	0	0	0	

and Children with no Clinical or Roentgenographic Evidence of Tuberculosis

Family History of Tuberculosis	Direct Exposure to Tuberculosis	Examination of Gastric Washings for Tubercle Bacilli: Smear of Sediment	Production of Tuberculosis in Guinea-Pig	Method of Inoculation Into Guinea-Pig	Follow-up: Reexamination of Gastric Washings	Follow-up: Addenda
0	0	Negative	Positive	Intraperitoneal	.....	.....
0	0	Negative	Positive	Intraperitoneal	.....	Repeated examinations of sputum failed to demonstrate tubercle bacilli
0	0	Negative	Positive	Intraperitoneal	6 months later and also 1 year later, inoculation of gastric washings into guinea-pig did not produce tuberculosis	Child gained 6 pounds (2.7 Kg.) during 6 months; clinically well; new roentgenogram revealed Ghon focus in upper lobe of left lung and more calcification in lung roots; tonsillectomy done; no evidence of tuberculosis on histologic examination of tonsils
(Maternal aunt) +	+	Negative	Positive	Subcutaneous	.....	.....
(Maternal aunt) +	+	Negative	Positive	Subcutaneous	5 months later inoculation of gastric washings into guinea-pig did not produce tuberculosis	Tonsillectomy on second admission after 5 month interval; histologic examination of tonsils revealed discrete and confluent tubercles
0	0	Positive; (tubercle bacilli demonstrated on direct smear of sediment)	....	....	7 months later inoculation of gastric washings into guinea-pig did not produce tuberculosis; smear of sediment gave negative reaction for tubercle bacilli	Preauricular node excised 1 week after tubercle bacilli had been demonstrated in gastric contents; histologic examination revealed caseous degeneration and tubercles in lymph node; tonsillectomy 7 months later; histologic examination did not reveal any evidence of tuberculosis in the tonsils
0	0	Positive; (tubercle bacilli demonstrated on direct smear of sediment)	....	....	2 months later inoculation of gastric washings into guinea-pig did not produce tuberculosis in the animal; smear of sediment gave negative reaction for tubercle bacilli	.....

in Which Inoculation of the Gastric Washings Into a Guinea-Pig Produced Atypical Tuberculosis

Family History of Tuberculosis	Direct Exposure to Tuberculosis	Examination of Gastric Washings for Tubercle Bacilli: Smear of Sediment	Production of Tuberculosis in Guinea-Pig	Method of Inoculation Into Guinea-Pig	Pathologic Changes in Guinea-Pig at Necropsy	Follow-up
(Father) +	+	Negative	Atypical	Intraperitoneal	Enlarged spleen; macroscopic whitish tubercle-like lesions in liver and spleen; enlarged peritoneal lymph nodes	.....
+	+	Negative	Atypical	Intraperitoneal	Gross appearance suggestive of tuberculosis; scattered miliary abscesses in liver; few tubercle-like structures in liver and spleen	On reexamination 4 months later, inoculation of gastric washings into guinea-pig did not produce tuberculosis
0	0	Negative	Atypical	Intraperitoneal	Gross appearance of spleen indicative of tuberculosis, but while microscopic examination revealed areas in liver and spleen suggestive of tuberculosis, no definite tubercles were seen	.....

in the case of an infant reported on by Ligner.[14] Poulsen and Andersen stated in their report that they knew of two similar but unreported instances. Furthermore, the finding of tubercle bacilli in the gastric contents is indicative of the presence of an active unhealed tuberculous focus even if roentgenographic and physical examinations give negative results, as shown in 7 of the cases reported here. In such cases the patient requires active therapy and must be regarded as a potential source of infection for uninfected children with negative cutaneous reactions to tuberculin. Finally, the procedure furnishes the pediatrician with an extremely reliable index as to how long treatment must be continued, for as long as tubercle bacilli can be demonstrated in the gastric washings, the tuberculous process must be considered to be active.

The proof of healing of the pulmonic process, in the opinion of both Poulsen and Andersen [10] and of Opitz,[6] is repeated negative tests of gastric washings. They expressed the belief that negative results of bacteriologic examination of the gastric washings for tubercle bacilli by the inoculation of guinea-pigs were far more decisive for children than any evidence that might be afforded by auscultation, roentgenographic examination, temperature curve and weight in determining the complete healing or the persistence of the active tuberculous process. Poulsen and Andersen [10] examined a number of children by gastric lavage at intervals of one or two months until negative results at two successive examinations were obtained. They found that in 7 cases tubercle bacilli were discharged for four or five months; in 21 cases, for from six months to one year; in 4 cases, for from one year to eighteen months, and in 1 case, for two years. These observers stated that in some cases, although auscultation may show normal conditions and roentgenographic examination may no longer reveal any infiltration, the discharge of tubercle bacilli may go on for a long time despite the clinical appearance of well-being and the absence of fever.

Of the 7 patients in this series of children with positive cutaneous reactions to tuberculin but with no roentgenographic or physical evidence of tuberculosis in whose gastric contents tubercle bacilli had been demonstrated, 4 were reexamined (table 2). No evidence of tuberculosis was found in the inoculated animal after an interval of six months, five months, seven months and two months, respectively. In 1 of the 3 cases in which results were suspiciously positive the patient was reexamined four months later, and the results were negative (table 3). Apparently the previously active tuberculous focus had healed in the interim, and no more bacilli were discharged.

14. Ligner, J.: Zum Studium der präallergischen Periode der Tuberkulose (Bacillennachweis), Ztschr. f. Kinderh. **50**:505-508, 1930.

It is of interest that in 2 cases, in which the patient was found to harbor tubercle bacilli in the gastric contents and was found to be free from bacilli on reexamination after an interval of six and seven months, respectively, tonsillectomy was done at the same time as the second test; in neither instance was any evidence of tuberculosis observed on histologic examination of the tonsils (cases 3 and 6, table 2). In a similar case, reexamination of the gastric contents after a five month interval by the inoculation of a guinea-pig gave negative results for tubercle bacilli, but after tonsillectomy, at the time of the second test, histologic evidence of tuberculosis of the tonsils was found (case 5, table 2).

CONCLUSIONS

The finding of tubercle bacilli in the gastric contents, as determined by the production of tuberculosis in a guinea-pig inoculated with the sediment obtained from the gastric washings, is indicative of the presence of an active tuberculous focus. This lesion is usually either in the lung or in a hilar or tracheobronchial lymph node which has ulcerated into the bronchus.

Tubercle bacilli were demonstrated in the gastric washings in 7 (8.14 per cent) of a series of 86 infants and children with positive cutaneous reactions to tuberculin in whom no recognizable evidence of tuberculosis could be demonstrated on physical or roentgenographic examination.

Examination of the gastric washings for tubercle bacilli by the inoculation of a guinea-pig with the sediment should be done for all infants and children with positive cutaneous reactions to tuberculin even if no evidence of tuberculosis has been found on physical and roentgenographic examination. The presence of tubercle bacilli in 8.14 per cent of the children in our series in the absence of physical and roentgenographic findings indicates the necessity of such a procedure, since the diagnosis of active tuberculosis could not have been made in these cases without the positive evidence obtained by examination of the gastric contents. These patients would have remained an unrecognized source of tuberculous infection to uninfected children with negative cutaneous reactions to tuberculin.

The pathologic studies reported on in this article were done by Dr. Perry J. Manheims.

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SIMILARITY OF ERYTHROBLASTIC ANEMIA AND CHRONIC OR CONGENITAL MALARIA

SUCCESSFUL TREATMENT OF EIGHT PATIENTS WITH QUININE

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In studying the blood from patients with erythroblastic anemia and those with chronic malaria, we were impressed by the great similarity of the findings in the two conditions. The anemia seems to be of the same type and reaches similar levels in cases of both diseases. The erythrocytes are diminished to an equal degree, and the relation of hemoglobin to the number of red blood cells remains undisturbed in patients suffering from either condition, even though at times there is an unappreciable degree of hypochromia. In neither disease is the poikilocytosis, anisocytosis or polychromasia marked. Likewise the appearance of nucleated erythrocytes, Jolly bodies (nuclear rests?) and basophilic granulation is observed with a corresponding degree of frequency in the two conditions. The fragility of the red blood cells is increased both in erythroblastic anemia and in chronic malaria, especially during the subfebrile and afebrile periods. In both diseases there is a characteristic increase in the percentage of lymphocytes. If one looks further into the pathologico-anatomic findings in the spleen and the bone marrow, a close similarity in the two conditions is evident, even in the face of the conflicting ideas of authors who have studied the spleen and the bone marrow of patients with erythroblastic anemia. All these laboratory observations suggested to us the existence of a similar causative agent for the two diseases, and this possibility was further supported by certain clinical observations.

In visiting the wards of the hospitals and the various clinics in Athens, during his recent visit to Greece, one of us (S. N.) was impressed by the following facts: Megalosplenic anemia, in the absence of any demonstrable pathogenic agent, was classified as erythroblastic anemia, and the patients were left more or less to their fate. The presence of the characteristic changes in the bones usually caused such a diagnosis to be the more readily established and discouraged any further investigation. However, when the persistence of the hospital intern proved the presence of the Laveran parasite in the blood and quinine was administered, prompt improvement followed.

Of particular interest were the cases of two infants, 10 and 11 months old respectively, lying side by side and both appearing equally ill, pale and emaciated. They had the same degree of moderate fever, equally enlarged spleens and livers and almost identical blood findings. The intern informed the visitor that at first both were classified as having erythroblastic anemia, but that after repeated examinations of the blood resulted in the demonstration of the Laveran plasmodium in one patient the diagnosis of his disease was changed to malaria. Quinine was then given him, and his symptoms began to improve. The fever subsided almost completely. The anemia was also gradually improving, with the reticulocytes and the nucleated red blood cells beginning to disappear, when the patients were seen by one of us. At his suggestion quinine was given the second patient also. Although repeated examinations, both before and after the administration of quinine, failed to reveal the presence of the Laveran parasite in the latter child, nevertheless his condition, like that of the first child, was improved and to the same extent.

This incident, especially, strengthened the idea that probably in many cases in which the diagnosis of erythroblastic anemia is made the condition may actually be a form of malarial infection, occurring during intra-uterine life or in some instances during the earliest days of infancy. To the theory of congenital malaria conforms also the fact that erythroblastic anemia, although at first observed and most extensively studied in America, is restricted to children of Mediterranean parentage, namely, Italian, Greek and Syrian. That malaria can be transmitted from the mother to the child is well known, and there is no necessity to dwell on the fact at present. It is likewise known that patients with chronic malaria can live for many years without showing any symptoms and that later, after a provocation of some sort, they may exhibit a flare-up of the disease. With these facts in mind, it will not appear illogical to assume that expatriated persons, harboring the Laveran parasite but without exhibiting any symptoms of malaria, often may transmit the disease to their children. That not all their children will develop the disease requires no argument. As is the case with congenital syphilis, malaria is not expected to affect all the children of the parasite-carrying mother. However, whether a child, acquiring the parasites from his mother and not showing any sign of the disease, may in turn transmit it to his children will have for the present to remain a matter of speculation. It is commonly accepted that this situation exists in the case of syphilis, although of course some doubt its possibility.

That the characteristic changes in the bones noted in erythroblastic anemia are not peculiar to that condition is recognized by most authors. First among them were Cooley and his associates, who stated that such changes "are the result of the reaction of the marrow to prolonged

overstimulation as a consequence of chronic hemolysis, beginning before the cortex is too firm to permit the overgrowth of marrow," [1] and who said that Grulee and Gänsslen had seen these changes in hemolytic conditions other than erythroblastic anemia. Of the most recent observers, Baty, discussing the case reported by Parker, stated the belief that the disease was "a disturbance of hematopoiesis, primarily involving the bone marrow" and that "the x-ray changes are simply secondary to a hyperplastic, actively growing marrow in a young individual whose bones are not entirely fixed." [2] The view expressed by this author is generally shared by other observers who have written on erythroblastic anemia. Whipple and Bradford,[3] however, have stated the opinion that the changes are not due simply to hyperplasia of the marrow, because alterations of the same nature are not seen in other types of anemia with extreme hyperplasia, and were inclined to attribute them to a metabolic defect, "racial or inherited," "associated with ductless gland imbalance." The racial factor seemed to impress greatly these and other authors, despite the fact that Syrians and Italians have little in common ethnologically. Lucia and Lucia,[4] in an excellent review of the subject, with a report of forty-five cases (of "splenic anemia . . . irrespective of . . . a concomitant diagnosis of luetic or tuberculous infection"), reached the conclusion that the condition is due to an inherited defective constitution. This defect, they said, is established in the ancestors of the patients, and they mentioned a number of diseases as possible agents: "syphilis, tuberculosis, sepsis, metabolic disturbances, possibly malaria, and other chronic illnesses." In common with other authors, they classified this condition among the leukemias, believing the changes in the red blood cells to be secondary to a reaction of the white blood cell-forming elements.

Aside from our views as to the etiologic factor involved in erythroblastic anemia, there is no warrant, we believe, for its classification among the leukemias. Such mild leukocytic reactions as characterize the disease are common to many septic processes, and the few immature white blood cells found in the peripheral blood are not uncommon in any condition in which marked hematopoietic activity of the bone marrow

1. Cooley, T. B.; Witwer, E. R., and Lee, P.: Anemia in Children with Splenomegaly and Peculiar Changes in Bones, Am. J. Dis. Child. **34**:347 (Sept.) 1927.

2. Parker, D. W.: Erythroblastic Anemia: Case Report, New England J. Med. **208**:1147 (June 1) 1933.

3. Whipple, G. H., and Bradford, W. L.: Racial or Familial Anemia of Children, Associated with Fundamental Disturbances of Bone and Pigment Metabolism (Cooley-Von Jaksch), Am. J. Dis. Child. **44**:336 (Aug.) 1932.

4. Lucia, S. P., and Lucia, E. L.: Splenomegalic Erythro-Leukemic Myelosis of Children in Relation to Chronic Infections, Univ. California Publ., Pub. Health **1**:337, 1933.

takes place. Identical immature forms of leukocytes of the myelocytic series are also found in erythremia (polycythaemia vera), a disease which, whatever its cause may be, is easily distinguished from the leukemias.

If the assumption that erythroblastic anemia is due to malaria is correct, the question why the former disease is limited to people of Mediterranean origin, since malaria is endemic in other countries as well, is not, we feel, difficult to answer. This type of anemia has been identified only recently, after intensive antimalarial campaigns had been made in countries more advanced in problems of public health than are those bordering the Mediterranean. Moreover, people in advanced countries are expected to receive adequate treatment when they contract a disease against which there is available, in the form of quinine, an almost infallible remedy. However, the various types of parasite found in different localities might be the most important factor in this connection. The different reactions of various races to the same stimulus should also be considered, though not exactly from the point of view of race but rather from the point of view of conditioned or modified resistance, developed among peoples who have borne the ravages of malaria for thousands of years. One of us (S. N.) is inclined to believe that sickle cell anemia of Negroes, which recently has been observed also among Greek families living in America, is probably in many cases a form of congenital malaria, and since his return to America he is making a study of the disease on that theory. The results of that study will be published later.

Having found that all the considerations and observations mentioned tended to confirm the idea that probably erythroblastic anemia is often a form of malarial infection, we decided, as a therapeutic test, to administer quinine to a number of patients under the observation of one of us (G. S.). Just at the beginning of this study the political disturbances in Greece forced one of us (S. N.) to leave that country. For this reason all the laboratory and clinical findings here reported are based on the observations of the other of us, who is likewise responsible for the dosage and mode of administration of the antimalarial agent used.

COMMON SYMPTOMS, PROCEDURES AND FINDINGS

Eight children ranging in age from 12 months to 12 years were treated with quinine. In the interest of economy of space and to avoid repetition, all common symptoms, procedures of examination and findings are given here, instead of in the individual case reports. Quinine, alternated weekly with atabrine, was given to two patients (cases 1 and 5). All the children had the peculiar cranial formation and facial expression, as well as the characteristic roentgenographic appearance of the bones, found in erythroblastic anemia. In one patient (case 1) these findings were not marked. The reaction to the Wassermann test was negative in all the

children. Seven children gave a negative reaction to the Pirquet-Mantoux test and one (case 5) gave a positive reaction. However, the patient whose reaction was positive, presented no other signs of active tuberculosis. Poikilocytosis and anisocytosis were observed in all cases and corresponded in severity to the degree of anemia present, subsiding when the red blood cell count became normal. In all cases repeated examinations were carried out to demonstrate the presence of the Laveran parasite in the peripheral blood. Almost all known methods were utilized, and the search was renewed at every opportunity. In two patients (cases 2 and 6) Laveran parasites of the malignant tertian type were found before treatment was commenced. In another patient (case 5) a shower of malignant tertian rings appeared suddenly on the thirty-second day of treatment, coinciding with a maximum rise in the number of eosinophils (18 per cent). After four days the parasites disappeared, and the eosinophil count also decreased. Seven patients were clinically and hematologically apparently cured. One patient (case 2) showed remarkable improvement while taking quinine; however, after twenty days of treatment the patient was removed from our care, and later we were informed that he went into relapse and died in about two months.

REPORT OF CASES

CASE 1.—A boy 5 years old was the youngest of ten children, of whom only five were living. One of the children who had died was "always very pale." The parents had observed a year previously that the patient began to look pale and to lose ambition and appetite. No definite information concerning fever was obtainable; however, the child had always been "anemic."

Examination and Findings.—The child was apparently well nourished. The skull was almost normal, and the circumference of the head was 51 cm. The height was 100 cm. (average normal, 104 cm.), and the weight, 14.1 Kg. (average normal, 18 Kg.). The spleen measured 3 cm. below the costal margin in the midaxillary line, and the liver, 2 cm. The hemoglobin content was 37 per cent (Sahli). Red blood cells numbered 2,230,000 per cubic millimeter; reticulocytes, 12 per cent; nucleated red blood cells,[5] 24 per hundred white blood cells; platelets, 86,000 per cubic millimeter; white blood cells,[6] 11,000 per cubic millimeter; young polymorphonuclears, 7 per cent; polymorphonuclears, 50 per cent; eosinophils, 4 per cent; lymphocytes, 36 per cent, and monocytes, 3 per cent. The bilirubin content was 0.15 mg. per hundred cubic centimeters of blood (average normal, 0.05 mg. per hundred cubic centimeters).

Treatment and Progress.—Quinine and atabrine, alternated weekly, were given by mouth. After one month the boy's general condition was improved considerably. The spleen and liver were barely palpable. The hemoglobin content was 70 per cent (Sahli). Red blood cells numbered 4,300,000 per cubic millimeter; reticulocytes, 2 per cent; nucleated red blood cells, 4 per hundred white blood cells; platelets, 180,000 per cubic millimeter, and white blood cells, 7,400 per cubic millimeter. The bilirubin content was 0.06 mg. per hundred cubic centimeters.

The child continued taking quinine and atabrine daily, alternating every week. When again seen, one and one half months later, he was enjoying complete health. The hemoglobin content was 75 per cent (Sahli). Red blood cells numbered

5. The term "nucleated red blood cell" is used throughout the paper, irrespective of the stage of maturity of the cell.

6. The number of nucleated red blood cells is not deducted from the number of white blood cells. This is true throughout the paper.

4,600,000 per cubic millimeter; reticulocytes, 2 to 3 per cent; nucleated red blood cells, none, and white blood cells, 6,200 per cubic millimeter.

CASE 2.—A boy 14 months of age was the fourth child in his family. The other three had died of "anemia" before they reached the second year of life. When the patient was 2 months of age the parents noticed that he was becoming "anemic" and losing his energy and appetite and that his spleen was becoming enlarged.

Examination and Findings.—The child was badly nourished and ill developed. There was accentuation of the parietal and frontal prominences (fig. 1). The circumference of the head was 42 cm. The height was 65 cm. (average normal, 76 cm.), and the weight, 6.1 Kg. (average normal, 10.5 Kg.). The spleen measured 5 cm. below the costal margin, and the liver, 3 cm. The hemoglobin content was 24 per cent (Sahli). Red blood cells numbered 1,910,000 per cubic millimeter; reticulocytes, 20 per cent; nucleated red blood cells, 40 per hundred white blood cells; platelets, 82,000 per cubic millimeter; white blood cells, 22,000 per cubic millimeter; myeloblasts, 2 per cent; myelocytes, 2 per cent; young polymorphonuclears, 4 per cent; polymorphonuclears, 32 per cent; eosinophils, 2 per cent, and lymphocytes, 58 per cent. The bilirubin content was 0.18 mg. per hundred cubic

Fig. 1 (case 2).—Note the groove between the parietal prominences and also the marked frontal prominence.

centimeters. After long and careful search, rare rings of malignant tertian Laveran parasites were found.

Treatment and Progress.—Quinine ethylcarbonate was administered daily by mouth. Twenty days after treatment was begun the patient's appearance and appetite were improved, and he had gained 2 Kg. in weight. The hemoglobin content was 48 per cent (Sahli). Red blood cells numbered 3,100,000 per cubic millimeter; reticulocytes, 8 per cent; nucleated red blood cells, 14 per hundred while blood cells; platelets, 89,500 per cubic millimeter; white blood cells, 12,000 per cubic millimeter; young polymorphonuclears, 6 per cent; polymorphonuclears, 40 per cent; eosinophils, 3 per cent; lymphocytes, 50 per cent, and monocytes, 1 per cent. The bilirubin content was still above normal, being 0.1 per hundred cubic centimeters. No Laveran parasites were found.

The mother took the child home, and we did not see him again. We were informed that treatment was abandoned with his removal from our care and that he died two months later.

This case is of interest in that the mother suffered from chronic malaria. Her spleen measured 10 cm. below the costal margin.

CASE 3.—A girl 12 months old was the youngest child of a mother who had had eight earlier pregnancies. Only five children were living. One of the children had died at 2 years of age from "anemia." There had been three miscarriages—one in the seventh month of pregnancy, one in the first month and the third resulting in a stillbirth at term. The patient was breast fed until she was 9 months old.

The mother had noticed that the child since she was 5 months of age had become pale, losing her appetite and failed to gain weight. At times she had a temperature as high as 38 C. (100.4 F.).

Examination and Findings.—The skull looked almost normal; however, the facial expression was typical (fig. 2). The height was 72 cm. and the weight, 7 Kg. (average normal, 10.2 Kg.). The spleen measured 2 cm. below the costal margin, and the liver, 3 cm. The hemoglobin content was 50 per cent (Sahli). Red blood cells numbered 3,100,000 per cubic millimeter; reticulocytes, 10 per cent; red blood cells with Jolly bodies, 16 per hundred white blood cells; nucleated red blood cells, 12 per hundred white blood cells; platelets, 100,000 per cubic millimeter; white blood cells, 19,000 per cubic millimeter; myelocytes, 2 per cent; metamyelocytes, 2 per cent; young polymorphonuclears, 5 per cent; polymorphonuclears, 32 per cent; eosinophils, 2 per cent; lymphocytes, 55 per cent, and monocytes, 2 per cent. The bilirubin content was 0.1 mg. per hundred cubic centimeters.

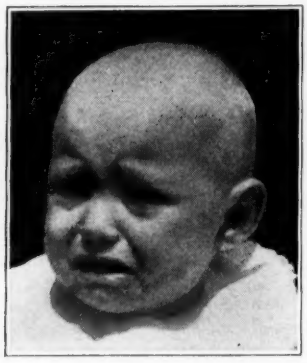

Fig. 2 (case 3).—The face is typical on account of the prominent cheek-bones.

Treatment and Progress.—The child was given quinine ethylcarbonate by mouth daily for two months. At the end of this period she manifested considerable improvement. The general condition and nutrition appeared excellent, and the spleen and liver were no longer palpable. The hemoglobin content was 75 per cent (Sahli). Red blood cells numbered 4,200,000 per cubic millimeter; nucleated red blood cells, none; platelets, 250,000 per cubic millimeter, and white blood cells, 8,200 per cubic millimeter. The bilirubin content was normal.

CASE 4.—A boy 18 months old was an only child and had been breast fed. The mother was badly nourished and looked anemic. Ten days after the child's birth the mother noticed that he was pale. Since he had a slight fever, the mother gave him quinine for a few days, which seemed to end the fever, without, however, satisfying the mother that he was entirely well. When 5 months old, the child had diarrhea, and the paleness became more pronounced.

Examination and Findings.—There was great accentuation of the parietal and frontal prominences (fig. 3). The circumference of the head was 49.5 cm. The height was 72 cm. (average normal, 79 cm.), and the weight, 9 Kg. (average

normal, 10.5 Kg). The spleen measured 3 cm. below the costal margin, and the liver, 1 cm. The hemoglobin content was 40 per cent (Sahli). Red blood cells numbered 3,500,000 per cubic millimeter; reticulocytes, 9 per cent; nucleated red blood cells, 17 per hundred white blood cells; red blood cells with Jolly bodies, 6 per hundred white blood cells; platelets, 140,000 per cubic millimeter; white blood cells, 16,400 per cubic millimeter; myelocytes, 8 per cent; metamyelocytes, 7 per cent; polymorphonuclears, 39 per cent; eosinophils, 1 per cent; lymphocytes, 43 per cent, and monocytes, 2 per cent. The bilirubin content was 0.29 mg. per hundred cubic centimeters.

Treatment and Progress.—The patient was given quinine by mouth for two months. At the end of this period considerable improvement was noticed. The spleen and liver were barely palpable. The hemoglobin content was 70 per cent (Sahli). Red blood cells numbered 4,300,000 per cubic millimeter; reticulocytes, 1 per cent; nucleated red blood cells, 1 to 2 per hundred white blood cells; platelets, 250,000 per cubic millimeter, and white blood cells, 10,400 per cubic millimeter. The bilirubin content was still high, being 0.13 mg. per hundred cubic centimeters.

Fig. 3 (case 4.)—Although the child is only 18 months old, the bony changes are marked.

CASE 5.—A girl 5 years old was the third child in her family. One child had died of "anemia"; the other was living and apparently well. When 2 years of age the patient began to look pale and emaciated, and occasionally had temperatures ranging between 37.5 and 38 C. (99.5 and 100.4 F.).

Examination and Findings.—The child looked pale and ill nourished. Her height was 96 cm. (average normal, 103 cm.), and weight, 11.1 Kg. (average normal, 17 Kg.). There was marked prominence of the frontal, occipital and especially the parietal bones (fig. 4). The spleen measured 6 cm. below the costal margin, and the liver, 3 cm. The Pirquet-Mantoux test gave a positive result, but no clinical symptoms of tuberculosis were present. The hemoglobin content was 23 per cent (Sahli). Red blood cells numbered 1,460,000 per cubic millimeter; reticulocytes, 16 per cent; red blood cells with Jolly bodies, 8 per hundred white blood cells; nucleated red blood cells, 7 per hundred white blood cells; platelets, 114,000 per cubic millimeter; white blood cells, 16,400 per cubic millimeter; metamyelocytes, 3 per cent; young polymorphonuclears, 5 per cent; polymorphonuclears, 38 per cent; eosinophils, 4 per cent; lymphocytes, 48 per cent, and monocytes, 2 per cent. The bilirubin content was 0.18 mg. per hundred cubic centimeters.

Laveran parasites were not found, although extensive examinations were repeated over many days.

Treatment and Progress.—Quinine and atabrine, alternating every week, were given by mouth. There was marked improvement of the general condition at the end of one month, and during that time the patient gained 2 Kg. in weight. On the thirty-second day of treatment the hemoglobin content was 56 per cent (Sahli). Red blood cells numbered 3,130,000 per cubic millimeter; nucleated red blood cells, 2 per hundred white blood cells; white blood cells, 14,000 per cubic millimeter; metamyelocytes, 3 per cent; polymorphonuclears, 45 per cent; eosinophils, 18 per cent; lymphocytes, 32 per cent, and monocytes, 2 per cent.

At the same time rings of malignant tertian Laveran parasites were found in considerable numbers. The presence of the parasites coincided with the peak of the eosinophil count, 18 per cent. The parasites continued to be found in the blood for four days, after which they disappeared completely. Quinine was continued for

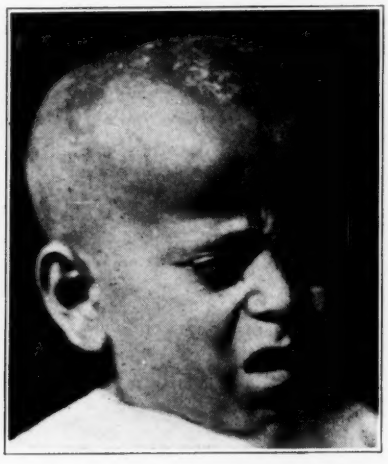

Fig. 4 (case 5).—Note the frontal prominences and also the typical expression of the face.

another month. The eosinophilia decreased gradually until the count was between 4 and 6 per cent. During this period repeated examinations for Laveran parasites failed to prove their presence. At the end of the third month of treatment the blood was found normal, and the child looked entirely well.

CASE 6.—A boy 16 months old was the third child in his family. Toward the end of the pregnancy the mother had had fever, which was diagnosed by a physician as malaria. The other two children were apparently normal. The patient had been breast fed, and he had looked well until he was 6 months old, when the mother noticed that he began to look pale and to lose energy. Gradually a slight fever was noted, ranging from 37 to 37.5 C. (99.5 F.). A physician found enlargement of the spleen and, without any further examination to establish definitely the diagnosis of malaria, prescribed quinine, which had been given, off and on.

Examination and Findings.—The child was 75 cm. in height (average normal, 85 cm.) and weighed 9.5 Kg. The frontal and parietal bones were slightly but definitely prominent (fig. 5). The circumference of the head was 49 cm. The spleen

measured 4 cm. below the costal margin, and the liver, 2 cm. The hemoglobin content was 50 per cent (Sahli). Red blood cells numbered 2,890,000 per cubic millimeter; reticulocytes, 5 per cent; red blood cells with Jolly bodies, 4 per hundred white blood cells; nucleated red blood cells, 8 per hundred white blood cells; platelets, 85,000 per cubic millimeter; white blood cells, 14,600 per cubic millimeter; metamyelocytes, 3 per cent; young polymorphonuclears, 5 per cent; polymorphonuclears, 38 per cent; eosinophils, 2 per cent; lymphocytes, 50 per cent, and monocytes, 2 per cent. The bilirubin content was 0.1 mg. per hundred cubic centimeters. Malignant tertian Laveran parasites were found.

Treatment and Progress.—Quinine was given by mouth daily for two months. The patient gained 3 Kg. in weight, and the liver and spleen were barely palpable at the end of this period. The hemoglobin content was 73 per cent (Sahli). Red blood cells numbered 4,200,000 per cubic millimeter; reticulocytes, 1 per cent; nucleated red blood cells, none; white blood cells, 8,200 per cubic millimeter; metamyelocytes, 1 per cent; young polymorphonuclears, 6 per cent; polymorpho-

Fig. 5 (case 6).—The prominences of the frontal and the parietal bones are typical but not marked, since the child is only 16 months old.

nuclears, 38 per cent; eosinophils, 1 per cent; lymphocytes, 52 per cent, and monocytes, 2 per cent. The bilirubin content was 0.06 mg. per hundred cubic centimeters.

CASE 7.—A boy 12 years old was the fifth of seven children, one of whom died when 18 months old of "anemia." One abortion, in the fifth month, had preceded the birth of the patient. The mother looked pale, and she stated that she suffered repeatedly from malaria. The child always had looked "anemic"; however, the mother had noticed during the last three years that he had become much paler and that his temperature ranged between 37 and 37.8 C. (98.6 and 100 F.). His appetite was considered fairly good.

Examination and Findings.—The bones of the head, especially the parietal and occipital, showed characteristic prominences (fig. 6). The height was 118 cm. (average normal, 140 cm.), and the weight, 26.2 Kg. (average normal, 35.2 Kg.). The spleen measured 2.5 cm. below the costal margin, and the liver, 1.5 cm. The hemoglobin content was 58 per cent (Sahli). Red blood cells numbered 3,320,000 per cubic millimeter; reticulocytes, 3 per cent; nucleated red blood cells, 7 per

hundred white blood cells; white blood cells, 18,200 per cubic millimeter; young polymorphonuclears, 6 per cent; polymorphonuclears, 42 per cent; eosinophils, 1 per cent; lymphocytes, 45 per cent; monocytes, 5 per cent, and plasma cells, 1 per cent. The bilirubin content was 0.15 mg. per hundred cubic centimeters.

Treatment and Progress.—The patient was given quinine by mouth daily. After one month's treatment, considerable improvement was noted, and he had gained 2 Kg. in weight. The hemoglobin content was 69 per cent (Sahli). Red blood cells numbered 4,100,000 per cubic millimeter; reticulocytes, 1 per cent; nucleated red blood cells, 2 per hundred white blood cells, and white blood cells, 10,800 per cubic millimeter. The bilirubin content was 0.12 mg. per hundred cubic centimeters. The spleen and liver still were enlarged. The boy was seen again thirty days later —ten days after he had discontinued taking quinine. The spleen and liver were then barely palpable. The hemoglobin content was 75 per cent (Sahli). Red blood cells numbered 4,800,000 and white blood cells 8,460 per cubic millimeter. The bilirubin content was 0.08 mg. per hundred cubic centimeters. The taking of quinine was to be resumed.

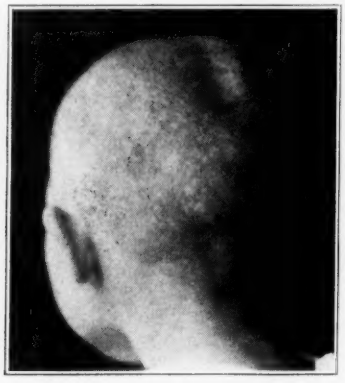

Fig. 6 (case 7).—Marked prominence of the occipital and the parietal bones.

CASE 8.—A girl 2 years old was the third living child in her family. The other two children were apparently well. There had been two abortions, in the third and the sixth month. Both parents suffered from chronic malaria, having typical paroxysms from time to time. The patient had been breast fed. When she was 8 months of age icterus developed after an attack of diarrhea, accompanied with fever, which lasted for three days. Since that time the child had begun looking pale.

Examination and Findings.—The girl was 71 cm. in height (average normal, 84 cm.) and weighed 8.5 Kg. (average normal, 12.2 Kg.). There was marked prominence of the parietal and frontal bones (fig. 7). The circumference of the head was 48 cm. The spleen measured 4 cm. below the costal margin, and the liver, 1 cm. The hemoglobin content was 47 per cent (Sahli). Red blood cells numbered 2,760,000 per cubic millimeter; reticulocytes, 15 per cent; red blood cells with Jolly bodies, 8 per hundred white blood cells; nucleated red blood cells, 24 per hundred white blood cells; platelets, 118,000 per cubic millimeter; white blood cells, 12,600 per cubic millimeter; myelocytes, 3 per cent; young polymorphonuclears, 7 per cent; polymorphonuclears, 33 per cent; eosinophils, 3 per cent;

lymphocytes, 52 per cent, and monocytes, 2 per cent. The bilirubin content was 0.185 mg. per hundred cubic centimeters.

Treatment and Progress.—She was given quinine ethylcarbonate by mouth daily. Within fifteen days the condition improved markedly. The spleen at the end of that time measured 2 cm. below the costal margin, and there was a gain in weight of 1.3 Kg. The hemoglobin content was 60 per cent (Sahli). Red blood cells numbered 3,640,000 per cubic millimeter; nucleated red blood cells, 15 per hundred white blood cells, and white blood cells, 10,000 per cubic millimeter. The bilirubin content was 0.175 mg. per hundred cubic centimeters.

The child continued to take quinine ethylcarbonate for another thirty days, and her condition was further improved. The weight increased to 11.5 Kg., the spleen was only 1 cm. below the costal margin, and the liver was barely palpable. The hemoglobin content was 77 per cent (Sahli). Red blood cells numbered 4,380,000 per cubic millimeter; reticulocytes, 2 per cent; nucleated red blood cells, 1 to 2 per hundred white blood cells; platelets, 235,000 per cubic millimeter; white blood cells, 7,000 per cubic millimeter; myelocytes, 1 per cent; young polymorphonuclears, 5 per cent; polymorphonuclears, 44 per cent; lymphocytes, 48 per cent, and monocytes, 2 per cent. The bilirubin content was 0.06 mg. per hundred cubic centimeters.

Fig. 7 (case 8).—Typical facial expression and prominence of the frontal and the parietal bones.

SUMMARY AND CONCLUSIONS

Eight children presenting all the clinical symptoms and hematologic findings commonly observed in erythroblastic anemia were treated, as a therapeutic test, with quinine. In two cases Laveran parasites of the malignant tertian type were found before treatment was commenced, and in one case such parasites appeared during the progress of treatment.

Seven of the children were apparently completely relieved of all their clinical and hematologic symptoms within a maximum period of three months. The other child showed remarkable improvement after administration of quinine for twenty days, but treatment was then abandoned, and the child died two months later.

It is assumed that erythroblastic anemia, in certain cases at least, is a form of malaria, most probably congenital.

The belief is expressed that in certain cases sickle cell anemia may likewise be another form of congenital malaria. Investigation of this subject is being carried on by one of us (S. N.), and the results will be reported later.

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Case Reports

SCURVY WITH AN UNUSUAL SYMPTOM

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AND

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PHILADELPHIA

ROENTGENOGRAPHIC REPORT BY

HERMAN W. OSTRUM, M.D.

This case of scurvy is reported because the patient presented an unusual symptom—the sudden appearance of a subaponeurotic hemorrhage which involved the left posterolateral aspect of the head.

REPORT OF A CASE

J. W., a Negro girl aged 10 months, was admitted to the Philadelphia General Hospital on Aug. 11, 1936. The family and the social history were irrelevant. Both parents were working irregularly. Five other children were said to be living and well.

Past Medical History.—The child was born prematurely at six and one-half months. At birth she weighed 3 pounds (1,307 Gm.). She was artificially fed barley water, Karo corn syrup and evaporated milk. During the winter she received 9 drops of cod liver oil daily. She did not receive orange juice or tomato juice. At 3 months of age she began to lose weight. She was placed in a hospital for three months and was discharged therefrom in good condition. Afterward she gained weight, but she received no prophylactic treatment of any kind.

Present Illness.—Four days before her admission to the Philadelphia General Hospital her mother first noticed a soft lump on the left side of the child's head. During the two or three days just before we saw the infant she had ceased making attempts to stand erect. The right leg had seemed tender to the touch. Anorexia and irritability, without gastro-intestinal symptoms, had appeared. Repeated careful questioning of both parents failed to elicit any history of a fall or other trauma which might account for the swelling of the child's head, nor was there any external evidence of such trauma.

Examination.—On her admission to the hospital the infant weighed 15 pounds and 1 ounce (6,832 Gm.). The rectal temperature was 100 F., the pulse rate 140 and the respiratory rate 55. The patient appeared somewhat undernourished but not acutely ill. She lay in bed listlessly but became fretful when approached.

The frontal eminences of the head were prominent. A soft, fluctuant, uniform swelling was noted on the left side of the scalp (fig. 1 *A*). It extended from the sagittal suture to just above the left ear. In the anteroposterior direction the

From Pediatric Service No. 1 and the Department of Radiology of the Philadelphia General Hospital.

swelling covered the posterior left half of the head. The floor of the swelling could be palpated and apparently was bone, the continuity of which seemed to be intact, and at the edges of the swelling the bony floor appeared to shade into and be continuous with the neighboring bones of the skull. There were no expansile pulsations over the swelling. The occiput was flattened, and the fontanels were not patent. The ears, the eyes and the nose were normal. The conjunctivae were not jaundiced.

Examination of the mouth revealed that two lower central incisors had erupted. No hemorrhagic or ecchymotic areas involving the hard or the soft palate, the buccal surfaces or the gums were noted. Of especial interest was the normal appearance of the gums surrounding the two lower central incisors. The tonsils were relatively large and injected. The pharynx was red, and there was a postnasal drip. The buccal cavity was otherwise normal.

Examination of the neck showed moderate adenopathy.

The chest was flat. A rachitic rosary, Harrison's groove, a wide subcostal angle, flaring rib margins and indentation of the xyphoid cartilage were noted.

The lungs, the heart, the abdomen and the genitalia were normal, and no abnormal neurologic reflexes were present.

No unusual tenderness affecting especially the right thigh, such as was described by the mother, was noted. No swellings were present in the extremities, except that there was enlargement of the radial epiphyses. There was no limitation of motion in the limbs.

Results of Laboratory Studies.—The urine was normal. The hemoglobin content of the blood was 9 Gm. per hundred cubic centimeters, or 52 per cent. The blood count showed 3,430,000 red cells and 13,300 white cells, of which 38 per cent were polymorphonuclears, 58 per cent lymphocytes and 4 per cent monocytes. There were 250,000 platelets. The bleeding time was three minutes and the clotting time six minutes. The fragility test showed partial hemolysis with a 0.5 per cent solution of sodium chloride and complete hemolysis with a 0.34 per cent solution. The Kahn reaction was negative. Chemical examination of the blood showed calcium 10.4 mg. and phosphorus 5.9 mg. per hundred cubic centimeters. Fluid obtained by aspiration from the swelling was not sufficient in quantity to make possible a test of its specific gravity. It was found to contain 1,330,000 red cells and 1,700 white cells, of which 45 per cent were polymorphonuclears, 30 per cent lymphocytes and 5 per cent endothelial cells. Culture and smear of the fluid showed that it was free from all organisms, including tubercle bacilli. The reaction to the Mantoux test with 0.0002 mg. of tuberculin and with 0.005 mg. of tuberculin, given intracutaneously, was negative.

Diagnostic Impressions and Possibilities.—Clinical rickets was recognized. The age of the child (10 months), the history of the sudden recent appearance of the swelling and the absence of expansile pulsation at once excluded the possibility of injury at birth or of a congenital defect of the skull which would have permitted herniation of the contents (as in neonatal cephalohematoma, meningocele and meningo-encephalocele).

There were no external evidences of trauma over the site of the swelling or elsewhere, and the parents were definitely positive in their statements that the child had received no injury. She showed no pupillary disturbances, local palsy, or abnormal neurologic reflexes, and although she was listless her mind was clear and she became irritable when she was approached or handled. There was no bleeding from the ears or nose, nor was there any escape of cerebrospinal fluid. A roentgenogram of the skull gave no evidence of fracture. It was recognized that

fracture of the skull would have accounted readily for the swelling, even in the presence of some dyscrasia of the blood, such as scurvy. Hence the need for establishing the presence or the absence of fracture became doubly important.

The possibilities mentioned having been excluded, the necessity for determining the nature of the fluctuant mass seemed pressing. Puncture was made. The absence of pus at once excluded the possibility of infection. The fluid was recognized as blood, macroscopically and by laboratory study. The low fever and the moderate leukocytosis could be explained by absorption of the fibrin content of the extravasation. The exclusion of trauma and of fracture as the cause of the bloody tumor made it necessary for us to consider some dyscrasia of the blood, such as scurvy, thrombocytopenic purpura or leukemia, as the possible etiologic factor. Jaundice was absent. Studies of the blood excluded all the diseases mentioned except scurvy. The facts stated in the history—that the child had received no protective substance (orange juice or tomato juice) and that she exhibited sensitiveness of the right leg and change in disposition (irritability) and refused to attempt to stand erect—made the diagnosis of scurvy strongly probable, although absence of hemorrhagic manifestations in the gums around the erupted teeth is extremely rare in cases of florid scurvy. The absence of pain, tenderness and swelling along the femur and the absence of limitation of motion, seen in the light of the subsequent roentgenographic findings, also are unusual in this type of case. It is worthy of special emphasis that the hematoma of the right femur was not discovered roentgenographically until August 29, that swelling of this part was never noted clinically and that the hematoma of the skull appeared on August 7, four days before the admission of the patient to the hospital and hence twenty-two days before the hematoma of the femur was discovered. The diagnosis of scurvy was confirmed by roentgenograms of the long bones and by the subsequent history of the case, including the patient's response to antiscorbutic treatment.

Clinical Course.—The rectal temperature ranged between normal and 102.2 F. between August 11 and 19. After that period it remained normal.

On August 14 the swelling seemed to be larger and the overlying skin tense. Twenty-five cubic centimeters of bloody fluid was aspirated. It did not coagulate, although the laboratory reported it to be blood. The swelling continued to increase in size until August 19, when it seemed less tense and somewhat smaller. By August 25 it had practically subsided, although the contour of the head was not entirely symmetrical. The remaining elevation was, however, no longer fluctuant but felt hard and bonelike. During its subsidence the mass had gradually lost its fluctuant feel, and when pressure was exerted the examiner experienced a sensation as of a separation in a semisolid substance. This was interpreted as being due to an organizing blood clot. On September 30 there was no longer any clinical evidence of this abnormal condition (fig. 1*B*).

Treatment.—In addition to a diet suitable to her caloric needs and digestive capacity the patient received 3 ounces (90 cc.) of pure orange juice daily. On August 29 an equal amount of tomato juice was substituted for the orange juice. In addition, a preparation of concentrated vitamin D was administered, and until the fever had left, a simple mixture was given to combat it.

Roentgenographic Report.—On Aug. 12, 1936, roentgenograms of the long bones and of the skull were taken. The long bones showed generalized atrophy, with thinning of the cortices and sclerotic dense irregular lines in the metaphysial plates. There were slight cupping and flaring of the ends of the bones, caused by associated rickets. The centers of ossification were visible, showing concentric rings of bony density (Wimberger's ring). Spur formations, most marked in

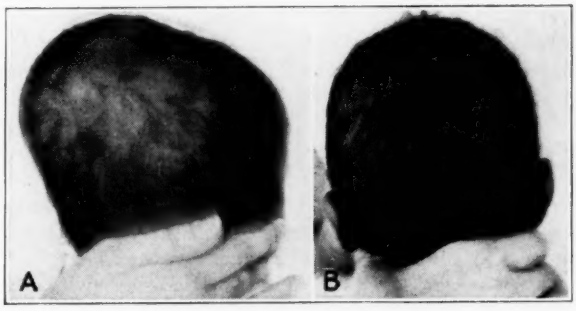

Fig. 1.—Photographs of the head of the patient. *A*, taken on her admission to the hospital, shows a large fluctuant swelling occupying the left posterolateral aspect of the head, and *B*, taken just before her discharge from the hospital, shows that the swelling has completely disappeared and that the head has returned to its normal symmetrical contour.

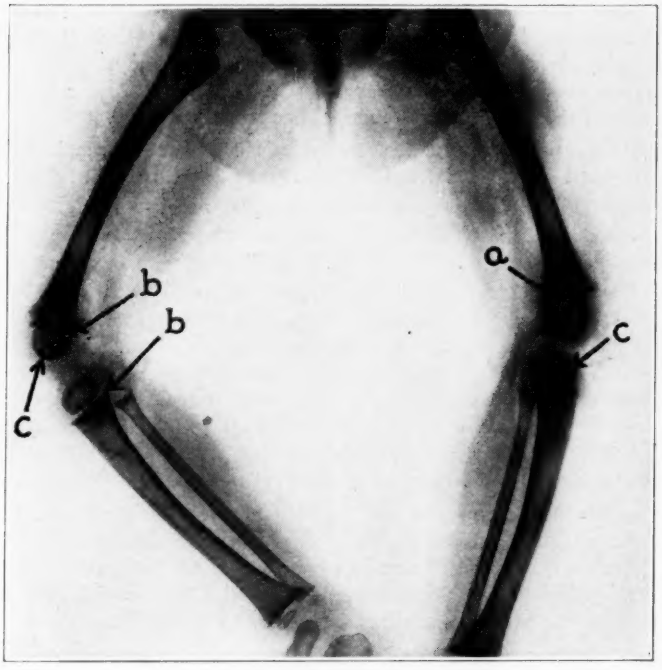

Fig. 2.—Roentgenogram of the long bones, taken on Aug. 12, 1936, showing a dense sclerotic zone in the metaphyses (*a*), spurring (*b b*), dense concentric rings (Wimberger's rings) in the centers of ossification (*c c*) and generalized atrophy of the bones and thinning of the cortices.

the tibia and the fibula, were clearly demonstrated (fig. 2). The anterior ends of the ribs showed bulbous expansion and clubbing (scorbutic rosary).

The frontoparietal region on the left side of the skull showed a dense area of soft tissue, well circumscribed and sharply defined. It was seen well above the external plate of bone and was due to subaponeurotic hematoma. There was no evident involvement of the bony structures, and the absence of fracture was definitely noted.

On August 17 two roentgenograms of the skull were taken. The tumor of soft tissue on the left side of the head in the parietal region was seen to better advantage from the lateral tangential point of view. The bones were not involved, and there was no evident increase or decrease in the swelling (fig. 3).

In a roentgenogram of the long bones taken on August 28, there was for the first time evidence, in the right femur, of a subperiosteal hematoma, containing a considerable calcium deposit. The long bones and their epiphyses showed considerable improvement; there was less decalcification of the bones and there was a loss

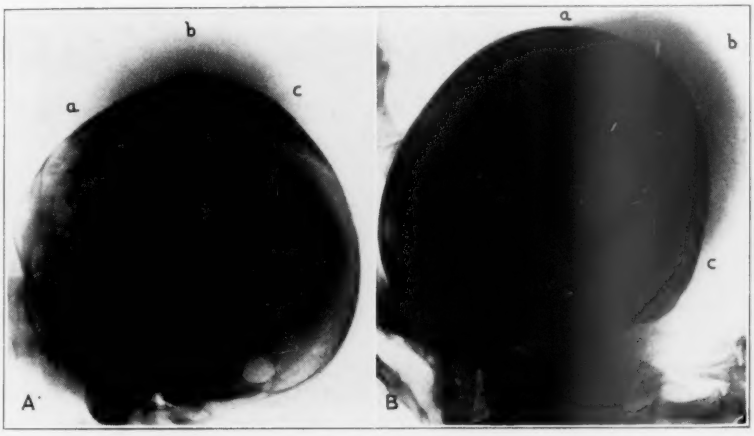

Fig. 3.—Roentgenograms of the skull taken on Aug. 17, 1936. *A*, the tangential left oblique view, shows swelling of the soft tissue in the left parietal region due to the subaponeurotic hematoma (*a b c*), with no evidence of fracture of the bones. *B*, the tangential postero-anterior view, shows the subaponeurotic hematoma (*a b c*) in the left parietal region, with no involvement of the internal or the external plates of the skull.

of the sharply defined zone in the metaphysial plates because it was being replaced by uniformly dense bone (fig. 4 *A*).

On September 18 the long bones gave additional roentgenographic evidence of healing; there was some absorption of the calcified hematoma of the right femur. Only a small amount of new bone was seen along the lower portions of the shaft. There was more calcification as well as less atrophy of the bone, and the typically dense zones in the ends of the bones were being replaced by uniformly dense areas of newly formed bone (fig. 4 *B*).

A roentgenogram of the skull, taken on September 24, showed the subaponeurotic hematoma to be much smaller. Deposits of new bone in the soft tissues paralleling the external plate were seen. There was no involvement of the bony structures (fig. 5).

Fig. 4.—Roentgenograms of the long bones. *A*, taken on Aug. 28, 1936, shows a hematoma (*a b*) in the right femur (visualized for the first time because of the beginning of healing and of calcification in the hematoma), recalcification of the remaining bones, denser rings, the epiphysial centers (*c c*) and the absence of atrophy. *B*, taken on Sept. 18, 1936, shows evidence of healing in the gradual absorption of the calcified hematoma (*a*) of the right femur.

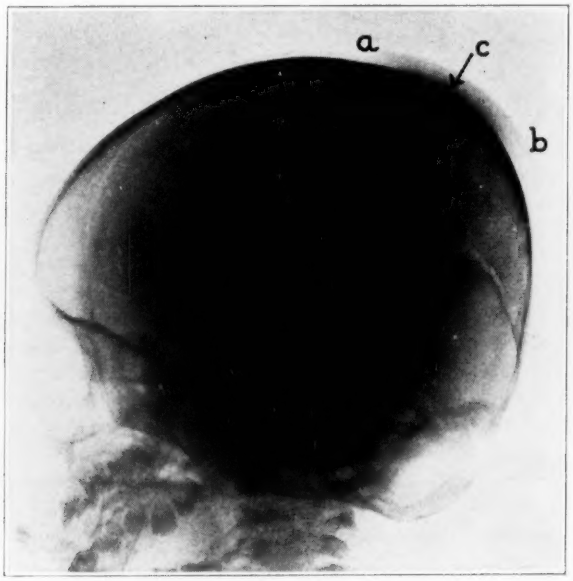

Fig. 5.—Roentgenogram of the skull, taken on Sept. 24, 1936, showing that the subaponeurotic hematoma (*a b*) is much smaller and that new bone (*c*) has been deposited in the soft tissues paralleling the external plate. There is no involvement of the bony structures.

COMMENT

Gilman and Tanzer [1] reported a case of subdural hematoma in an infant with scurvy and in addition provided an excellent bibliographic review. They referred to a case of intracranial hemorrhage due to scurvy reported by Willis in 1668, a case of hemorrhagic pachymeningitis reported by Hayem in 1871, a case of intracranial hemorrhage associated with scurvy—probably the first in the literature—reported by Sutherland in 1894 and a case of a large blood clot on the superior surface of the brain associated with subcortical hemorrhage also reported by Sutherland. In 1894, according to these authors, Ord reported a case of a large subdural hemorrhage associated with scurvy, and in 1896 Meyer described a case in which pachymeningitis haemorrhagica interna was accompanied with scurvy. They referred also to a case of subdural hemorrhage reported in 1919 by Sammis; to three cases of extradural bleeding with scurvy, one reported by Möller in 1862 and one each reported by Sato and Nambu in 1908; to a case of pachymeningitis haemorrhagica interna reported by Glauber in 1928, and to a study made by Scherer in 1913 on thirteen adults between 20 and 25 years old with scurvy who came to autopsy, 25 per cent of whom showed various types of intracranial hemorrhage. In their complete bibliographic review they also included reference to the investigation of infantile scurvy made by the American Pediatric Society in 1898, in which three of twenty-nine deaths occurring in 379 patients with scurvy were recorded as due to cerebral hemorrhage. In addition, they included many other references to aberrant (kidney) and intracranial and intraspinal bleeding due to scurvy.

Dunnington [2] reported a case of exophthalmos due to scurvy in a 7 month old infant. One of us (H. L., Sr.) [3] saw a patient suffering from the same ailment and described the phenomenon as suddenly developing, usually unilateral exophthalmos, followed by ecchymosis of the lid.

Innumerable references may be found to the occurrence of hemorrhage in unusual and unexpected places giving rise to unexplained symptoms. Thus bleeding of the kidneys has been mentioned, and Hess [4] stated that hemorrhage in the lower right quadrant of the abdo-

1. Gilman, B. B., and Tanzer, R. C.: Subdural Hematoma in Infantile Scurvy: Report of Case with Review of Literature, J. A. M. A. **99**:989 (Sept. 17) 1932.

2. Dunnington, J. H.: Exophthalmos in Infantile Scurvy, Tr. Am. Ophth. Soc. **29**:37, 1931.

3. Lowenburg, Harry, Sr.: Infant Feeding and Allied Topics, Philadelphia, F. A. Davis Company, 1916, p. 285.

4. Hess, Alfred: Scurvy, Past and Present, Philadelphia, J. B. Lippincott Company, 1920, p. 84.

men gave rise to suspicion of appendicitis. He also referred to trauma, such as that caused by the undue pressure of diapers or clothes, as a factor of great importance in determining the location of the hemorrhage. Hence, even when there is a history of trauma, scurvy is not to be ruled out on that account alone.

Recently Scott [5] reported a case under the title "Nontraumatic Spontaneous Subaponeurotic Hematoma: Its Probable Relation to Atypical Scurvy." The patient was 14 years of age, and a roentgenogram of her long bones showed them to be normal. It may well be that the evidence of minute multiple fractures and spurring, emphasized as the earliest changes noted in the bones in infantile scurvy, does not necessarily appear in the older child or in the adult. The patient apparently was well established in the state of puberty. "The full effect of vitamin C therapy could not be ascertained . . . as the blood under the scalp was removed largely by aspiration." Scott mentioned two cases, similar to this one, which were reported by Aballí and Valledor.[6] In these, as well as in the case here reported, the hemorrhage occurred under the scalp, and the response to vitamin C therapy was prompt and convincing.

SUMMARY AND CONCLUSIONS

A case of scurvy is reported in which the first clinical manifestation was the sudden appearance of a subaponeurotic hemorrhage involving the left posterolateral aspect of the head.

There were no hemorrhagic manifestations in the gums, not even around the site of two erupted lower central incisor teeth.

Except for some tenderness on pressure, clinical evidences of subperiosteal hematoma of the right femur were absent throughout the entire duration of the condition. Certainly there was no swelling. Although it must have occurred earlier, the hematoma was discovered by roentgenogram for the first time twenty-two days after the appearance of the subaponeurotic hematoma of the head.

In cases of obscure (so-called subclinical) scurvy or atypical manifestations of scurvy, one may depend for early diagnosis on roentgenograms made and interpreted by a roentgenologist having specialized experience with the bones of infants and children.

325 South Seventeenth Street.

5. Scott, Michael: Nontraumatic Spontaneous Subaponeurotic Hematoma: Its Probable Relation to Atypical Scurvy, J. A. M. A. **107**:348 (Aug. 1) 1936.

6. Aballí, A., and Valledor, T.: Bol. Soc. cubana de pediat. **5**:201 (June-Dec.) 1933.

CRYPTOCOCCIC BRONCHOPNEUMONIA

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Cases are occasionally observed in the routine practice of the physician which are typical from the clinical standpoint but in which the laboratory findings are distinctly novel. Such cases are probably more frequent than a perusal of the medical literature would indicate, and they are therefore worth reporting.

The following is a report, with laboratory findings, of a case in which the condition appeared clinically to be bronchopneumonia but in which Cryptococcus was isolated and none of the well known incitants of pneumonitis were present.

REPORT OF A CASE

History.—H. P., a boy aged 10 years and 8 months, was well nourished and healthy. He had had measles, whooping cough, chickenpox and scarlet fever before he was 8 years old; his tonsils had been removed when he was 7, and he had had lobar pneumonia of six days' duration when he was 8, in 1933.

Clinical Course.—On Dec. 15, 1935, the patient had an infection of the upper respiratory tract and was kept in bed two days. On December 23 he still had a slight cough. In the afternoon of December 25 he had a chill lasting twenty minutes, and at 8 p. m. his temperature had risen to 103 F. He was first seen the morning of the twenty-sixth, at which time he had a severe pain on the left side in the lower part of the thoracic region. Examination revealed suppression of the breath sounds, bronchial breathing and diminished resonance in the lower left lobe and pleural friction rub in the axillary line. The child was markedly cyanosed; his respiratory rate was 36. A diagnosis of lobar pneumonia was made. The course of the pneumonia from the third to the fourteenth day is shown in figure 1.

On the fifth day of illness a second severe chill occurred, accompanied by extreme cyanosis and symptoms of collapse. Eight hours later the entire right lung showed extensive involvement. The cyanosis was so intense that the patient was placed in an oxygen tent, which greatly added to his comfort. Not until seven days later could he be removed from the tent without acquiring cyanosis and respiratory distress so marked that it was necessary to replace him in the tent. There was a marked nonproductive cough.

On the fifteenth day the temperature reached 99 F., and in the next eleven days it reached 100 F. every day. Until the twenty-fifth day there were many quiescent pneumonic areas. Resolution was slow.

Laboratory Observations.—Because no sputum was raised, material for cultures was taken from low in the pharynx, on the fifth day, so that the pneumococcus could be typed. The culture showed yeast organisms. The same results were obtained with cultures of material from the pharynx on the sixth and the eighth day. On the tenth day sputum was obtained which also showed yeast organisms. Two months after the patient's recovery, cultures of material taken from deep in the pharynx were negative for yeast organisms.

Examination of the blood on the sixth day showed 6,900 leukocytes, of which 60 per cent were polymorphonuclears. After the first blood transfusion the count showed 10,800 leukocytes, with 65 per cent polymorphonuclears. After the third

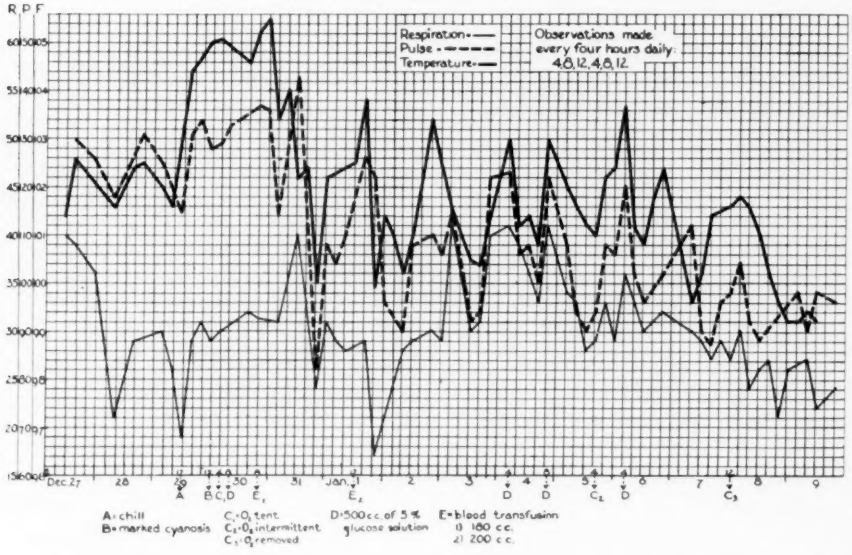

Fig. 1.—Chart showing the course of the pneumonia from the third to the fourteenth day.

transfusion the count showed 10,300 leukocytes, with 62 per cent polymorphonuclears.

Urinalysis gave negative results throughout the illness.

A roentgenogram was made on the tenth day to eliminate a suspicion of fluid in the right pleural cavity (fig. 2). This film showed extensive pneumonic involvement. The lower left lobe showed beginning resolution, while the right lung showed extensive bronchial pneumonia, particularly in the lower right lobe.

Investigation of the Yeast Organism.—No sputum could be obtained for culture, although the clinical symptoms at the onset were those of typical lobar pneumonia and the clinical course that of bronchopneumonia. Cultures of material taken from the throat were negative for both pneumococci and streptococci, showing mainly staphylococci. A culture of material taken from far back in

the throat, in the region of the nasopharynx, plated on Sabouraud's medium showed a heavy growth of budding yeast forms. This culture was transferred to 6 per cent honey agar.

Colonies appeared in forty-eight hours and were isolated for study. The colonies on honey agar were dull, round and creamy tan. No mycelia developed in corn meal agar, and ascospores could not be demonstrated by any of the usual methods, e. g., with corn meal agar or carrot plugs. Hence it was determined that the organism belonged to the Cryptococcus group (unicellular forms reproducing by budding which do not have mycelia or ascospores).

Microscopically, the organism was a small oval budding form measuring 2 or 3 microns in length and 1 or 2 microns in width, although some examples were spherical (fig. 3). No capsule could be demonstrated with india ink preparations or in animal tissues.

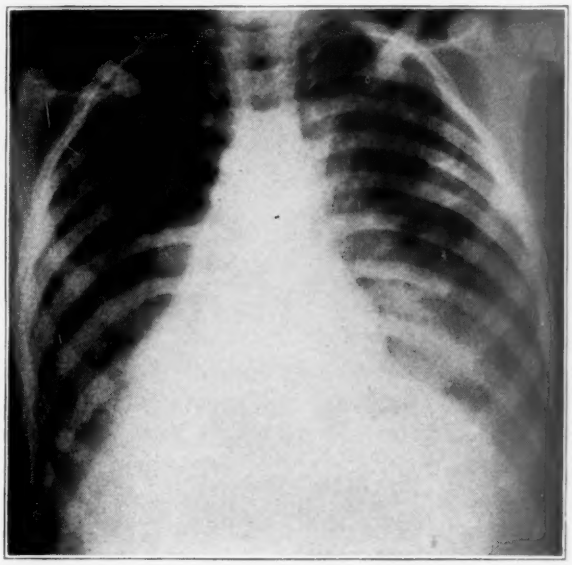

Fig. 2.—Roentgenogram of the chest made on the tenth day of the illness. It shows the extensive pneumonic involvement.

The organism was cultured in dextrose, maltose, sucrose, lactose, levulose, inulin, dextrin, galactose, raffinose and mannose. The specimens were allowed to incubate at room temperature for three weeks and were read each week. Acid and gas appeared in the dextrose, levulose and mannose; all the other cultures were negative. The organism did not liquefy gelatin. The giant colony (fig. 4) on honey agar measured about 1.8 cm. in diameter after a growth of four weeks, had a smooth surface and varied from tan in the center to cream around the slightly lobulated edges.

Inoculations into animals were employed to detect possible pathogenicity of the organism, as it did not morphologically resemble known pathogenic cryptococci (Cryptococcus hominis—Torula histolytica).

Cultures were grown for forty-eight hours on honey agar slants, and the growth was emulsified in 10 cc. of sterile saline solution. Two full grown rats

were inoculated intraperitoneally with 1.5 and 3 cc., respectively, of this suspension. Both animals were definitely ill for the first few days, after which they improved. One rat was killed in five days; the only notable gross pathologic change was contraction of the omentum, which contained a few hard nodules. All the organs contained yeast, as was demonstrated in culture. The second rat was killed

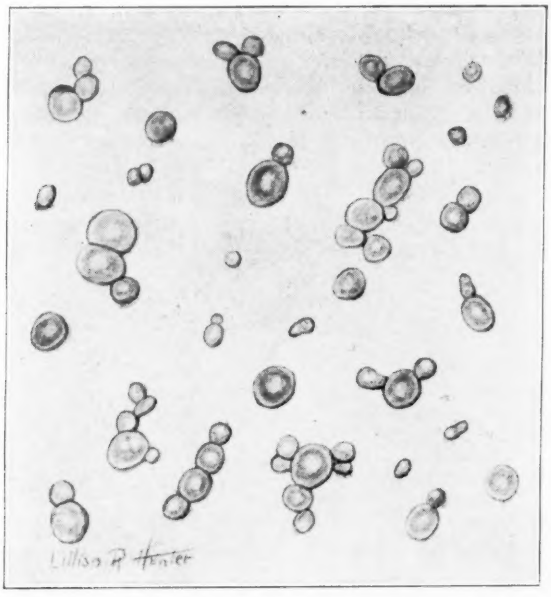

Fig. 3.—Drawing which shows the organism as it appeared microscopically.

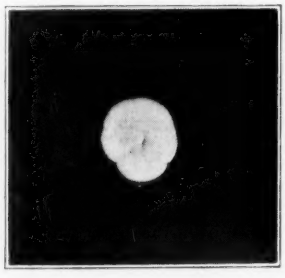

Fig. 4.—Photograph of a giant colony, two-thirds actual size.

in ten days and showed the same pathologic condition except that the adrenals were inflamed. Material for culture was taken from all the organs; that from the spleen, the omentum, the kidney and the adrenals and fluid from the pleural cavity were positive for cryptococci.

The culture recovered from these animals was inoculated into another rat, which was killed in three weeks. The omentum was contracted into a hard mass containing many hard nodules, which were filled with thick caseous material.

Cultures were made of material from all the organs, but only the peritoneal fluid and the material from the omentum and the kidney were positive for cryptococci.

The organism biochemically, morphologically and culturally resembled Cryptococcus glabratus, described by Anderson (1917). This organism was classified as Cryptococcus, group 1, by Benham (1933) and Fisher and Arnold (1936). Benham (1935) included Cryptococcus aggregatus and Cryptococcus ovoideus with C. glabratus in group 1. She found these types on 5 per cent of one hundred normal tongues investigated and on from 23 to 32 per cent of other normal cutaneous surfaces (Benham 1933). However, group 1 (Fisher and Arnold 1936), including only C. glabratus, was found in the mouth of only one of forty-eight normal children and in the mouth of only one of twenty-eight normal adults. It was not found on other normal cutaneous surfaces but did occur in the feces, gastric contents and vagina occasionally.

COMMENT

The rôle of certain types of cryptococci as disease-producing entities in human beings has been the subject for much investigation and dissension. Bronchomycosis due to a wide variety of fungi and yeasts has been reported in the literature. Castellani (1928) classified bronchomycosis as (1) types caused by the yeastlike fungi—Monilia, Cryptococcus, Saccharomyces, Blastomycoides and Endomyces, and (2) those induced by the filamentous fungi, such as Nocardia, Aspergillus, Penicillium and Sporotrichum. He classified bronchomoniliasis into two types, mild and severe, the former characterized by scanty mucopurulent expectoration, no fever and a few râles in the chest and the latter by hectic fever, emaciation, hemorrhagic expectoration, patches of dulness, fine crepitations and pleural rubbing in the chest, the roentgenogram in cases of the severe form resembling that in cases of tuberculosis. Bronchomycosis due to cryptococci presents a picture identical with that of bronchomoniliasis according to Castellani. He did not describe the type of cryptococcus, however. Stovall and Greeley (1928) reported a study of eighteen cases of primary bronchomycosis in which Monilia was isolated. Other cases of bronchomoniliasis have been reported by Marett (1933), Lewis (1933) and Bakst, Hazard and Foley (1934). The last-named authors included with their case reports a comprehensive review of the literature on bronchomoniliasis. They also stated that the clinical picture is variable in these cases.

Other cases of bronchomycosis have been reported as caused by T. histolytica. Busse (1895) isolated a yeast which he called Saccharomyces hominis from a woman with chronic pyemia accompanied by suppurative destruction of the skin, cornea, bones, lungs and spleen. He named the condition saccharomycosis (yeast infection). With Buschke (1895), he demonstrated its marked pathogenicity in white mice. The same organism was named C. hominis by Vuillemin. Curtis

(1896) described a yeast isolated from a sarcoma which he believed to be the etiologic factor because it produced tumors in rabbits; he named it Saccharomyces subcutaneus-tumefaciens. Von Hansemann (1905) described a yeast infection of the brain and meninges. Later, Stoddard and Cutler (1916) reported two cases of similar meningeal infection; they named their organism T. histolytica. Similar cases of torulosis or torula meningitis have been described by Shapiro and Neal (1925), Freeman and Weidman (1923) and Mitchell (1936). Another series of cases have been reported by Versé (1914), Brewer and Wood (1908) and Williams (1922), under the name of European blastomycosis. Benham (1934 and 1935) presented morphologic and immunologic evidence to show that all these strains are probably identical or at least closely related and should be known by the name of C. hominis.

In addition to meningitis, T. histolytica has been reported as the etiologic factor in pulmonary torulosis, both associated and unassociated with torula meningitis (Stoddard and Cutler [1916], Sheppe [1924], Berghausen [1927], Stone and Sturdivant [1929], Hirsch and Coleman [1929] and Hardaway and Crawford [1935]). It is interesting that Berghausen's patient raised no sputum at any time during the course of the disease, which terminated fatally, and that the organism was isolated from lesions of the tongue.

C. hominis (T. histolytica) is an accepted pathogen in spite of the fact that avirulent strains morphologically indistinguishable from the pathogen have been found on normal surfaces of the body by Benham (group III, 1935) and Fisher and Arnold (group II, 1936).

While the status of C. hominis as a factor in the production of disease is established, many other known species of cryptococci are reported as possible or doubtful pathogens. Greenbaum and Klauder (1922), in a survey of the skin of 150 persons, isolated a cryptococcus with which they could produce abscess formation in guinea-pigs. They also successfully produced a lesion in a human being on an abraded finger web with a cryptococcus. White (1927) reproduced lesions with cryptococci in the persons from whom they were obtained. Finnerud (1929) was able to reproduce perlèche successfully in human beings with cryptococci obtained from patients with perlèche. Similarly, Skolnik (1930) produced a lesion on a breast with a cryptococcus isolated from a perlèche lesion. White and Schwartz (1928) isolated cryptococci 31 times in 35 cases of eczema of the eyelids. In spite of a slight inflammatory reaction produced by experimental inoculation with cryptococci, Becker and Ritchie (1930) expressed doubt of their pathologic rôle in cutaneous infections.

No doubt many conditions have been reported in the literature as having been caused by various types of yeast when insufficient data had

been found to warrant the reporting of this fungus as the etiologic factor. Proof of the pathogenicity of little known species of cryptococci is believed to lie in their ability to produce disease in experimental animals. The majority of nonpathogenic cryptococci disappear quickly from the bodies of these animals after intraperitoneal injection and cannot be demonstrated in culture after two weeks.

The persistent pathogenicity in experimental animals of the cryptococci isolated from the throat of our patient, the initial toxic symptoms in the animals and the type of pathologic change observed at autopsy place this cryptococcus in the type of the potentially pathogenic strains. The absence of pneumococci and streptococci as demonstrable causative agents of the pneumonitis and the recovery of the patient add weight to the explanation that the cryptococcus was the etiologic factor. The case is as interesting to the mycologist as to the internist. This report emphasizes the fact that the mycologist observes the varying degrees of parasitism which are so well known to the bacteriologist. Yeasts and fungi cannot be classified as saprophytes and pathogens any more than can bacteria. Varying degrees of tissue-invading power are possessed by all parasitic forms of life.

SUMMARY

A case is presented of bronchopneumonia in a child in which there was no laboratory evidence of pathogenic bacteria in the nasopharynx or the sputum.

A yeastlike organism was found in three successive cultures from the pharynx and one from the sputum.

This organism was identified as C. glabratus.

The pathogenicity of this strain of C. glabratus for white rats was established.

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Progress in Pediatrics

IMMUNIZATION AGAINST SCARLET FEVER

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Scarlet fever is an acute infectious disease syndrome characterized by sore throat, a more or less typical enanthem, fever and a typical exanthem, or rash. The rash is the most characteristic feature, and by it alone can the diagnosis be confirmed. While this definition is generally accepted and while the majority of investigators consider scarlet fever a clinical entity, it must be kept in mind that competent, experienced clinicians, from Escherich to Schick, Hoyne, Dochez and Wadsworth, have not accepted it as a true entity or as due to a single, specific organism. Most of these dissenters have considered the rash and the other manifestations of the disease as allergic phenomena.

Since 1877, when Loeffler first observed streptococci in the pharynx of patients with scarlet fever,[1] more than thirty-four groups of investigators have announced the discovery of the etiologic agent of scarlet fever.[2] Eight of these isolated streptococci. Scarlet fever was experimentally transmitted to human beings by Miguel as early as 1833,[3] by Haywood in 1860,[4] by Stickler in 1883,[5] by Gabritschewsky in 1907 [6] and by the Dicks in 1921-1924.[7] No one can deny that typical scarlet fever may follow infection with streptococci from erysipelas, puerperal fever and other diseases and that, vice versa, these streptococcic diseases have followed infection from typical scarlet fever. Thus there are, as has been established by Gunn and Griffiths,[8] Okell,[9] Blake and Trask,[10]

From the Department of Pediatrics, Marquette University School of Medicine.

1. Loeffler: Mitth. a. d. k. Gsndhtsamte. **2**:421, 1882.

2. von Pfaundler, M., and Schlossmann, A.: Handbuch der Kinderheilkunde, ed. 4, Leipzig, F. C. W. Vogel, 1931, vol. 2, p. 108.

3. Miguel, M.: Lancet **2**:235, 1833.

4. Haywood, B., quoted by von Pfaundler and Schlossmann.[2]

5. Stickler, J. W.: M. Rec. **23**:316, 1883.

6. Gabritschewsky, G.: Zentralbl. f. Bakt. **41**:719, 1907; Berl. klin. Wchnschr. **44**:556, 1907.

7. Dick, G. F., and Dick, G. H.: Etiology of Scarlet Fever, J. A. M. A. **82**: 301 (Jan. 26) 1924; Experimental Scarlet Fever, ibid. **81**:1166 (Oct. 6) 1923; Experimental Inoculations in Scarlet Fever, ibid. **77**:782 (Sept. 3) 1921.

8. Gunn, W., and Griffith, F.: J. Hyg. **28**:250 (Dec.) 1928.

9. Okell, C. C.: Lancet **1**:815 (April 16) 1932.

(Footnotes continued on next page)

Dochez and Stevens,[11] Tunnicliff[12] and others, at least four serologic groups of hemolytic streptococci associated with scarlet fever. These four groups are not peculiar to scarlatina, and there is a close relationship between the various streptococcic diseases. E. C. Rosenow wrote that "specificity in scarlatinal hemolytic streptococci may be an acquired and temporary property."[13] A. B. Wadsworth, director of the Division of Laboratories and Research of the New York (state) Department of Health, stated:

> The experimental and clinical investigations of the etiology of scarlet fever have never established beyond question that Streptococcus haemolyticus is the primary incitant. The results of this work do, however, establish its practical importance from the therapeutic standpoint; that is, treatment with antistreptococcus serum is definitely indicated provided the serum neutralizes the toxin of the streptococcus involved in the infectious process, whether it is of a primary or of a secondary nature. There has been no demonstration of a specific streptococcus in relation to scarlet fever, erysipelas or any other form of streptococcic infection. . . . For the past six years we have worked intensively toward the selection of strains of Str. haemolyticus which would complement or broaden the valence and equal the antigenic potency of the Dochez, N. Y. 5, strain so that an effective vaccine and thus, also, an effective serum might be prepared for all forms of streptococcic infection. We have studied about 1,500 strains and are now hopeful that three, possibly four, of those selected will prove satisfactory.[13]

The rash of scarlet fever depends not only on the infection of the throat or wound with hemolytic streptococcus of sufficient virulence and the production of an adequate amount of toxin but also on the presence or absence of an adequate amount of antitoxin in the patient's circulation.

The cutaneous test which the Dicks have presented as a method to determine the susceptibility of persons to scarlet fever is a fairly reliable indication of cutaneous sensitivity to the Streptococcus scarlatinae filtrate toxin and a fairly reliable indication as to whether a person who contracts a streptococcic infection will also acquire a rash. Rosenow said that "immunity of the skin to the toxin does not necessarily mean immunity of the throat to infection by truly scarlatinal streptococci."[13] J. V. Cooke[13] and others[13] have voiced the same opinion. Wadsworth stated: "The diagnostic value of the intracutaneous test which was developed by the Drs. Dick is . . . limited to a determination of the susceptibility to the particular streptococcus toxins used in the test."[13]

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10. Blake, F. G.; Trask, J. D., Jr., and Lynch, J. F.: Observations on the Treatment of Scarlet Fever with Scarlatinal Antistreptococcic Serum: Preliminary Report, J. A. M. A. **82**:712 (March) 1) 1924. Blake, F. G., and Trask, J. D., Jr.: J. Exper. Med. **40**:381 (Sept.) 1924.

11. Stevens, F. A., and Dochez, A. R.: Proc. Soc. Exper. Biol. & Med. **21**:39, 1923.

12. Tunnicliff, Ruth: J. Infect. Dis. **49**:357 (Oct.) 1931.

13. Personal communication to the author.

The serious feature of scarlet fever is not the rash but the septic infection with the streptococcus. In studies on the presence of toxin (antigen) demonstrable in the circulation during clinical scarlet fever, Cooke found that "the amount of toxin had practically no relationship to the clinical severity of the toxemia, and surprisingly large amounts of toxin could at times be demonstrated in the serum in cases in which the condition was clinically mild." [13]

Gabritschewsky in 1907 prepared a vaccine from the killed scarlet fever streptococci and produced a symptom complex resembling scarlet fever in a few hours by subcutaneous injection.[6] His experiments have been repeated, chiefly by Russian and Polish physicians, and the results confirmed.[14] The most important result obtained seemed to be a prophylactic immunity of about two years' duration. This vaccine was available commercially in the United States until 1917 but did not become popular. The Dicks have prepared a bouillon culture filtrate of one strain of a scarlatinal hemolytic streptococcus, a scarlet fever toxin for immunization. The strength of this toxin is determined by the variable reaction to the cutaneous test. A total of 120,000 skin test doses is recommended for administration. The Dicks and their assistants maintained that there are no serious reactions to the injection of the prescribed doses of toxin. They also contended that a negative reaction to the toxin in a cutaneous test, which is produced in the majority of cases, means an immunity to scarlet fever which lasts for six years or more.

The Dicks have not disproved the contention of bacteriologists that the cutaneous reaction is only an indication of the sensitivity of the skin at that particular time to the toxin of that particular strain of organism. Bacteriologically, it is impossible to produce a lasting active immunity against any streptococcic disease. Admitting for the sake of argument the statistics of the Dicks and a few others which indicate that a negative reaction to a Dick test may be produced in 95 per cent of the persons inoculated with 120,000 skin test doses of the Dick scarlet fever toxin and granting, for the same reason, that a negative reaction to the cutaneous test may persist for several years, what does this prove? That the person who is subsequently infected with scarlet fever streptococci will not acquire a cutaneous rash. He may still acquire all the rest of the clinical picture of scarlet fever, including the most serious features of the disease, the sepsis and the toxemia, but his physician will be deprived of his sole criterion for diagnosis, the rash.

No one but the Dicks will deny that the injection of toxin in the prescribed doses often produces severe reactions. Witness the numerous articles in the literature suggesting modification of the original pro-

14. von Pfaundler and Schlossmann,[2] p. 113.

cedure for immunization.[15] Many of the reactions are more severe than the scarlet fever was in the majority of the cases we observed in Milwaukee in the recent epidemic (1934-1935).

No one but the Dick group maintains even that the reaction to the cutaneous test remains negative for six years or more. W. H. Park, director of laboratories for the New York (city) department of health, stated: "We do not use antiscarlatina serum as a preventive because scarlet fever at the present time is only moderately communicable and in the majority of cases is light." [13] He also said: "In two years after five injections of 75,000 or 100,000 skin test doses were given, the reaction in the majority of children with a strongly positive reaction to the Dick test had again become positive. We do not feel that we can rely on a longer immunization and therefore have not pushed the use of the vaccine actively in this city." [13] Wadsworth wrote:

> We have not favored active immunization against scarlet fever or any other form of streptococcic infection because the vaccines have not been prepared with a potency and valency adequate for all types and strains of Str. haemolyticus. Among the strains in common use, one in particular, the Dochez, N. Y. 5, has proved so much more effective antigenically that the addition of other strains constitutes a dilution. The Dick strains have not proved to be satisfactory antigens in our experience. There are certain situations in hospitals and institutions in which active immunization appears to be advisable, but in such instances it seems wise to use only the toxin of the Dochez, N. Y. 5, strain because of its broad antigenic valency. Usually, however, the time and the dose required for effective immunization render the procedure impracticable or unnecessary. In outbreaks, prompt treatment and the isolation of patients are the most important measures.[13]

Archibald L. Hoyne, director of the Municipal Contagious Disease Hospital of Chicago and clinical professor of medicine at the University of Chicago, stated:

> In institutions I think the use of Dick's scarlet fever toxin for active immunization is of utmost value in apparently preventing the development of scarlet fever. On the other hand, it does not prevent the development of sore throat at some future time in those who have been immunized. Moreover, in some instances it seems questionable whether those who acquire sore throat do not have scarlet fever without the rash.
>
> When private patients are being considered, I believe the problem of active immunization against scarlet fever with scarlet fever toxin is a much more difficult problem. Because many children have more or less severe reactions after one or two doses of the toxin, many of our Chicago physicians decline to adopt the procedure. Besides, many parents seem unwilling to take their children to a doctor's office on five separate occasions for the purpose of having injections made when a guarantee cannot be given that full protection is secured by such treatment.

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15. Rappaport, B.: Active Immunization to Scarlet Fever with Less Reaction, J. A. M. A. **106**:1076 (March 28) 1936. Robinson, J. H.: J. Immunol. **31**:373 (Nov.) 1936. Scarlet Fever Immunization, Queries and Minor Notes, J. A. M. A. **107**:1988 (Dec. 12) 1936.

Until further improvement is made in the method of active immunization against scarlet fever, it does not seem practicable to attempt extensive campaigns in schools or communities.[13]

The most widely quoted report of mass immunizations, one supervised by Dr. Gladys H. Dick, came from Kentucky. This report is quoted to support the value of immunization. In this report Jones stated:

Although this epidemic (Berea) promptly subsided within ten days following the initiation of this program (which included pasteurization of the milk), it is not our contention that the carrying out of these measures merits the entire credit for this accomplishment. *The outbreak was milk borne, explosive in type and, naturally, would have largely subsided following protection of the milk supply.*[16] [The italics are mine.]

There are other reports from Russia, Manchuria and Serbia.[17] On June 14, 1935, Henry reported the results of active immunization against scarlet fever in Philadelphia from 1932 to March 1, 1935.[18] His statement that "there have been no cases of sickness in those who have been declared immune or who have been immunized which are in any degree suggestive of scarlet fever without rash or of any other group of symptoms or complications of the disease" is open to challenge. In his article Henry described three cases in which scarlet fever developed in a so-called immunized child. Toomey, in a discussion of Henry's paper, listed five failures of protection in his series after five injections and stated that he had seen six other patients who contracted scarlet fever after having received the full five doses of toxin. He then said: "The type of scarlet fever present in the United States will have to be more severe for me to agree with Dr. Henry that this program should be made a public health measure."[18] The number of cases in which scarlet fever develops after a person has received injections of scarlet fever toxin will never be known because in most of them a typical rash will not develop—at least not for a few years.

In 1934 an epidemic of scarlet fever commenced in Milwaukee. During 1934 there were 5,965 cases, with 20 deaths, and in the first six months of 1935 there were 4,541 cases, with 24 deaths. The health commissioner, Dr. J. P. Koehler, began immunization by the Dick method in March 1934 and by Sept. 1, 1935, had immunized 10,500 children. One third of those who started to have the injections did

16. Jones, John L.: Newer Aspects in the Prevention and Control of Scarlet Fever, with a Report of Sixty Thousand Complete Immunizations, read before the American Public Health Association in Indianapolis on Oct. 11, 1933.

17. Thomson, D., and Thomson, R.: Ann. Pickett-Thomson Research Lab. **6**:244 (Dec.) 1930.

18. Henry, J. N.: A Study of Active Immunization Against Scarlet Fever in Charitable Institutions and Public Schools of Philadelphia, J. A. M. A. **105**:488 (Aug. 17) 1935.

not return to complete them. Of these, 57 per cent had reactions, and it is reported that 17 contracted scarlet fever.[19]

During this epidemic 95 per cent of the patients I saw in private practice and at the Milwaukee Children's Hospital had such a mild form of the disease that they required no treatment. The incidence of complications was negligible. Partly because of this, the state board of health shortened the quarantine period during this epidemic to twenty-one days. M. J. Fox[20] reported that of the patients sent to the city's isolation hospital, 83 per cent had the disease so lightly that they required nothing more than nursing care. Usually only the patients who have the most severe form of the disease are sent to the hospital. The patients in these hospitals and my private patients, therefore, had less discomfort from the scarlet fever than they would have experienced from five injections of the Dick toxin plus two cutaneous tests.

There is no object in arguing about statistics. They can be made to prove or disprove anything. Witness the controversy over BCG and the enormous number of statistics cited by the French. Without adequate controls and careful observation no conclusions are justified, particularly after mass immunizations with loose methods of supervision and control.

The Dicks advised against immunization in an epidemic because the first two or three injections lower the resistance of a person to scarlet fever. Any of the injections may produce a clinical picture indistinguishable from that of true scarlet fever, or true scarlet fever may develop in the course of injections, and the patient is allowed the freedom of the community while he spreads the disease.

Veldee, of the United States Public Health Service, in 1933 reported the production of a scarlet fever toxoid.[21] This preparation is given in three doses, which total 128,000 skin test doses. It has been given to several thousand persons, "with systemic reactions practically absent in the young and occurring only occasionally in the adult." Retests with raw toxin gave negative results in 87 per cent of the cases. This work deserves a great deal more consideration than it has received.

Boyd, of the Rockefeller Institute, in his recently published book, "Preventive Medicine," said:

> Susceptibles may be actively immunized with scarlet fever toxin. As yet a standard procedure for the purpose has not been devised. A method that would confer an effective immunity with not more than three immunizing doses would appear desirable. Toxin that has been neutralized by mixture with sodium ricinoleate appears most suitable at present.[22]

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19. Koehler, J. P.: Am. J. Pub. Health **25**:1359 (Dec.) 1935.
20. Fox, M. J.: Wisconsin M. J. **34**:19 (Jan.) 1935.
21. Veldee, M. V.: Pub. Health Rep. **48**:549 (May 25) 1933.
22. Boyd, Mark F.: Preventive Medicine, ed. 5, Philadelphia, W. B. Saunders Company, 1936, p. 83.

SUMMARY

May I, then, summarize my brief against the present method of immunization against scarlet fever with the following statements?

1. The bacteriologists and most clinicians do not accept a specific strain of the hemolytic streptococcus as the etiologic agent of scarlet fever.

2. The bacteriologists insist that it is not possible to produce an active immunity against any streptococcus.

3. The specificity of the Dick test has not been proved.

4. Active immunization with the Dick toxin or vaccine prevents the development of a rash for two or more years. If the inoculated person becomes infected with scarlet fever, his physician is robbed of his most valuable diagnostic sign.

5. The Committee on Clinical Investigation and Scientific Research of the American Academy of Pediatrics reported on June 11, 1934: "The whole matter of scarlet fever immunization, both by the toxin and by the toxoid methods, is being investigated . . . by the U. S. Public Health Service and the Massachusetts State Board of Health. The results of these investigations must be awaited before it is possible to evaluate their usefulness." [23] In April 1935 the Special Committee on Prophylactic Procedures Against Communicable Diseases of the American Academy of Pediatrics reported: "Active immunization [against scarlet fever] . . . is not recommended as a general public health procedure, since reactions both local and general are frequent . . . and for the reason that the degree and duration of immunity have not been definitely established." [24]

23. Talbot, F. B.: Report of the Committee on Clinical Investigation and Scientific Research of the American Academy of Pediatrics, J. Pediat. **5**:106 (July) 1934.

24. Report of Special Committee on Prophylactic Procedures Against Communicable Diseases of the American Academy of Pediatrics, J. Pediat. **6**:588 (April) 1935.

News and Comment

GENERAL

Physicians Certified by American Board of Pediatrics.—The following physicians have been certified by the American Board of Pediatrics since the last report:

Joseph Battaglia, Brooklyn; William H. Baughman, Oakland, Calif.; Julius Blechschmidt, Philadelphia; James E. Bowman, Philadelphia; Jacob Brem, Boston; Claudius Virgil Calvin, Bridgeport, Conn.; Paul Dennison Clark, Burlington, Vt.; Clement B. P. Cobb, New York; Moses Cooperstock, Marquette, Mich.; A. Mordecai Cowan, Cleveland; Albert Graham Davis, Utica, N. Y.; Charlotte Fisk, Madison, Wis.; Maurice M. Fliess, Clifton Forge, Va.; Harold J. Freedman, Boston; Fred S. Gachet, Rochester, N. Y.; Alice Lilian Arnold Gilbert, New York; Alcibiades A. Giroux, Duluth, Minn.; Elizabeth Ross Harrison, New Haven, Conn.; Robert Beebe Hauver, Cleveland; John Mark Higgins, Sayre, Pa.; Jeanette Hork, Chicago; Kenneth Gabriel Jennings, Brooklyn; Robert Emmett Jennings, East Orange, N. J.; Abram Kanof, Brooklyn; Paul Brooks Kreitz, Bethlehem, Pa.; Hugh Francis Leahy, Albany, N. Y.; Jacques M. Lewis, New York; Pascal Francis Lucchesi, Philadelphia; Claire Cutten Manwell, Northampton, Mass.; Theodore Melnick, Philadelphia; Isaac J. Muldawer, Philadelphia; Nelson Newmark, Springfield, Mass.; Leonard Parente, New Haven, Conn.; David Stewart Polk, Rosemont, Pa.; Donald Dunn Posson, Rochester, N. Y.; Thomas Ryan Quinn, Pittsburgh; Gabriel J. Rubin, Boston; Harry S. Snyderman, Philadelphia; Alfred A. Trivilino, Jamaica, N. Y.; Fred C. Turley, St. Louis; Joseph Weiner, New Haven, Conn.; Israel J. Wolf, Paterson, N. J.; Irving J. Wolman, Philadelphia.

Thirty-eight of these physicians were examined.

Advisory Board for Medical Specialties.—The annual meeting of the Advisory Board for Medical Specialties, which is the coordinating board of the twelve certifying boards in the various specialties, the Association of Medical Colleges, the American Hospital Association, the Federation of State Medical Boards of the United States, and the National Board of Medical Examiners, was held at Atlantic City, N. J., on June 6, 1937.

The following officers and members of the executive committee were elected: Willard C. Rappleye, M.D., president, New York; W. P. Wherry, M.D., vice president, Omaha; Paul Titus, M.D., secretary-treasurer, Pittsburgh; W. B. Lancaster, M.D., Boston, and R. C. Buerki, M.D., Madison, Wis.

Dr. Louis B. Wilson of Rochester, Minn., the retiring president of the board, was elected an emeritus member.

DEATHS

On Sunday, May 23, Dr. Gustave Lippmann, of St. Louis, died after a lingering illness.

Dr. Lippmann was one of the most respected and best beloved pediatricians in St. Louis, and his going will leave a gap which will be hard to fill. His geniality and conservative common sense were much appreciated by his colleagues.

Abstracts from Current Literature

Biochemistry; Bacteriology, and Pathology

PREVENTION OF INTRANASALLY INOCULATED ENCEPHALITIS (ST. LOUIS) TYPE IN MICE AND OF POLIOMYELITIS IN MONKEYS BY MEANS OF CHEMICALS INSTILLED INTO THE NOSTRILS. CHARLES ARMSTRONG and W. T. HARRISON, Pub. Health Rep. **51**:1105 (Aug. 14) 1936.

Solutions of picric acid buffered at a range of p_H which gave no coagulation of protein afforded no protection, but solutions buffered in an acid range that coagulated protein afforded protection in mice and monkeys. Buffered solutions with an acidity of p_H 2.8 or greater exert a protective influence but to a less degree than 0.5 per cent solutions of picric acid of the same acidity. Mixtures of picric acid with sodium aluminum sulfate in saline solution protected all of twenty monkeys against infection which occasioned poliomyelitis in sixteen of twenty nonprepared controls. Five-tenths per cent solutions of picric acid of p_H 4.4 and more acid buffers were also effective in mice and in a small group of monkeys.

SANFORD, Chicago.

SALMONELLA SUIPESTIFER VAR. AFRI. AUST. IN AN OUTBREAK OF FOOD-POISONING IN THE CAPE PENINSULA. E. C. GREENFIELD and H. M. JUDD, South African M. J. **10**:544 (Aug. 8) 1936.

A Salmonella associated with food poisoning is described. It attacks both arabinose and dulcitol, which Kolle and Hetsch stated is done only by exceptional strains of Salmonella suipestifer. By its general cultural characters it can be classified as Salmonella suipestifer; yet serologically, although it appears to be related to S. suipestifer, it is not identical with it. For this reason it is regarded as a special variety of S. suipestifer, and the name Salmonella suipestifer var. Afri. Aust. is proposed.

GONCE, Madison, Wis.

ARTHUS' PHENOMENON IN OROPHARYNGEAL MUCOSA. K. ARSLAN, Arch. ital. di otol. **48**:18 (Jan.) 1936.

Arslan succeeded in producing Arthus' phenomenon in the velum pendulum palati of rabbits by means of submucous velar injections of horse serum, repeated at regular intervals. The development of Arthus' phenomenon is slower in the oropharyngeal than in the submucous tissues of rabbits, and the oropharyngeal lesion is smaller than the subcutaneous lesion—probably because the reactivity of the submucous tissues to local sensitization is diminished as compared to that of the subcutaneous tissues. The velar histogenous reaction is different from that caused by chronic processes of inflammation or irritation of the mucosa due to stimulation and repeated trauma. The same experiments carried on in guinea-pigs failed to produce Arthus' phenomenon in the oral mucosa. The failure confirms the opinion that guinea-pigs are refractory to the development of reactions of local anaphylaxis. The success in producing Arthus' phenomenon in the oropharyngeal mucosa of rabbits shows that the mucosa of the upper part of the respiratory tract reacts to the heterogenous substances with which it comes in contact. This fact is of importance for further investigation of the relation between the upper part of the respiratory tract and the production of phenomena of humoral or histogenous anaphylaxis. The results of the experiments do not interfere with the nature of Arthus' phenomenon, which may be a reaction of local anaphylaxis or of a specific sensitivity of the tissues to heterogenous substances.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

Metabolism; Infant Feeding; Milk and Other Foods

STUDIES IN ABSORPTION OF UNDIGESTED PROTEINS IN HUMAN BEINGS: VI. THE ABSORPTION OF UNALTERED PROTEIN FROM THE ABNORMAL GASTRO-INTESTINAL TRACT. IRVING GRAY and MATTHEW WALZER, Am. J. Digest. Dis. & Nutrition **3**:403 (Aug.) 1936.

In this experiment forty subjects were used. These were human beings, all of whom had functional or organic disease of the gastro-intestinal tract. Test meals were carried out on each to ascertain the amount of free hydrochloric acid and the total acid content. These patients were then grouped as follows: Those persons were normal whose free hydrochloric acid was between 20 and 30 degrees. Hyperacidity was constant at 40 degrees or over, and the total acidity was constant over 60 degrees. Hypo-acidity was considered present when the free hydrochloric acid was constant at less than 10 degrees and the total acid was constant at less than 20 degrees. An acidity was considered present in persons in whom no free hydrochloric acid was present at any time.

Peanut was the protein chosen for this experiment. Peanut was excluded from the diet of the subject for twenty-four hours preceding the experiment. The subject was then passively and locally sensitized on the forearm by the intracutaneous injection of 0.05 cc. of a 1:10 dilution of serum which had been obtained from a patient who was extremely sensitive to peanut and who maintained a marked reaction to intracutaneous tests with this antigen in very high dilution. One or two days later the subject reported without breakfast for the peanut test meal. This consisted of a milk made by dissolving 10 Gm. of raw ground peanuts in 30 cc. of water to which was added 1 Gm. of sugar and 0.05 cc. of oil of cloves. Shortly after the ingestion of this meal a reaction developed at the sensitized site, which usually consisted of pruritis, erythema and the formation of a wheal. The onset of this reaction marked the entrance of the undigested peanut protein into the circulation.

The absorption time was measured from the time the test meal was taken to the time the first objective symptom was noted at the sensitized site, namely, erythema or a wheal.

All patients with a past, present or family history of hay fever, asthma or similar forms of atopy were excluded from this study.

The absorption time in persons with normal acidity averaged eighteen and seven-tenths minutes. The absorption time in persons with hyperacidity averaged twenty-seven and five-tenths minutes. The absorption time in persons with hypo-acidity averaged fifteen and nine-tenths minutes, and the absorption time in persons with anacidity averaged eleven and seven-tenths minutes.

The authors' conclusions, therefore, were that the rapidity with which unaltered protein is absorbed following its oral administration is directly influenced by the degree of gastric acidity. In patients with hyperacidity the absorption of unaltered protein is definitely retarded. In the presence of hypo-acidity the absorption is accelerated.

MORRISON, Savannah, Ga.

SKELETAL CHANGES IN THE RAT INDUCED BY A RATION EXTREMELY POOR IN INORGANIC SALTS. MIRIAM F. CLARKE, ALEXANDER L. BASSIN and ARTHUR H. SMITH, Am. J. Physiol. **115**:556 (May) 1936.

A progressive decrease in the ash content of the bones resulted. The bones continued to grow in length in spite of the stationary body weight, although less than noted in the controls. The breaking strengths of the bones of the rats poor in salts were from a third to a half of the corresponding values for controls of the same age. The breaking strength appears to be more closely correlated with the organic pattern as reflected in the size of the bone than with the proportion of inorganic salts. Following return to a proper diet the bones failed to grow normally, showing that permanent damage was produced by the nutritional adjustment employed.

NOURSE, Cleveland.

LACTATION IN ADRENALECTOMIZED RATS. ROBERT GAUNT and CHARLES E. TOBIN, Am. J. Physiol. **115**:588 (May) 1936.

No definite evidence was produced in support of the hypothesis that there is a second hormone in the adrenal cortex, separate from the life-maintaining hormone, which is essential for lactation. NOURSE, Cleveland.

INFLUENCE OF IRRADIATED ERGOSTEROL AND PARATHYROID EXTRACT UPON THE RATE OF DISAPPEARANCE OF INTRAVENOUSLY INJECTED CALCIUM CHLORIDE. SMITH FREEMAN, Am. J. Physiol. **115**:701 (May) 1936.

Viosterol and parathyroid extract act alike in prolonging the rate of removal of injected calcium from the blood of dogs. NOURSE, Cleveland.

CERTIFIED MILK FOR INFANT FEEDING. P. R. CASSIDY and J. A. TOBEY, Arch. Pediat. **53**:420 (June) 1936.

Cassidy and Tobey advocate the use of certified milk in the diet of all infants and children because of the sanitary and nutritional qualities of such milk.

ORR, Buffalo.

INDUCED HYPERCALCEMIA: ITS POSSIBLE THERAPEUTIC RELATION TO THROMBOCYTOPENIC PURPURA. H. LOWENBURG and T. M. GINSBERG, J. A. M. A. **106**: 1779 (May 23) 1936.

Another case of acute hypercalcemia produced by intentional overdosage with parathyroid extract occurred in a boy with thrombocytopenic purpura. The symptoms in man are similar to the toxic symptoms described in animals. They consist of vomiting followed by weakness, apathy and lethargy. Once hypercalcemia is established, the bleeding time is reduced, clot retraction occurs normally, and the platelet count is increased. These striking results suggest the possible value of inducing hypercalcemia in the treatment of thrombocytopenic purpura by large doses of parathyroid extract. No explanation is offered for the apparent cures. Calcium gluconate was used to protect the bones from withdrawal of calcium.

BONAR, Salt Lake City.

CALCIUM METABOLISM IN RELATION TO DISEASE. ELLA G. A. WEBB, Irish J. M. Sc., May 1936, p. 209.

Absorption of calcium depends on the acidity of the gastro-intestinal tract, the food it is given with and the amount of fat. Calcium is excreted by the kidneys but more especially by the large bowel. The soluble salt is excreted by the kidneys and the insoluble salt by the bowel. Thus excess phosphorus causes excretion in the bowel.

Webb discusses the relation of calcium, phosphorus, vitamin D and parathyroid hormone in bone formation. The theories concerning prolonged bleeding in obstructive jaundice are given. Spasmophilia is due to a decrease of calcium in the blood serum, caused by lack of vitamin D. Calcium is low in severe nephritis. Lead in the blood acts much the same as calcium.

Webb feels that there is a definite relation between the blood calcium, chilblains, urticaria and possibly other diseases of the skin. SNELLING, Toronto, Canada.

SOME FEATURES OF CALCIUM METABOLISM. WILFRID SHELDON, Irish J. M. Sc., May 1936, p. 220.

The body requires 0.5 Gm. of calcium daily, but the intake in the food must be around 2 Gm. to supply this. The fetus in the last three months stores 0.3 Gm. per day. Premature infants need vitamin D, calcium and phosphorus in large amounts in food. Milk is the main source of supply; 1 pint (473 cc.) contains

a little more than 1 Gm. Absorption is increased by acidity of the bowel and decreased if too much fat is fed. The adult body contains 1,400 Gm. of calcium, most of which is in the skeleton. The action of parathyroid extract and vitamin D appears to be similar, though vitamin D causes healing of rickets and parathyroid extract causes only great decalcification of the bones. The author quotes reported cases of hypervitaminosis D, in only two of which, reported by Hess, were cures proved to have been obtained. There is slight likelihood that a small overdosage of vitamin D does any harm.

SNELLING, Toronto, Canada.

THE THERAPEUTIC ACTION OF IRON. L. J. WITTS, Lancet **1**:1 (Jan. 4) 1936.

Feeding experiments with animals indicate that only about half the iron in food can be assimilated. The administration of iron to normal animals and human beings does not affect the blood count, but much of the iron may be retained in the body. The iron requirement in childhood has been estimated at 0.76 mg. per hundred calories of food ingested. Iron is administered internally in medicine for its hematinic action and for its tonic or roborant action. The soluble ferrous salts are the most active. The ideal preparation of iron still awaits discovery. Iron acts as a nutrient and not as a stimulant for the blood-forming organs.

LANGMANN, New York.

THE PROBLEM OF CALCIFICATIONS. F. S. HANSMAN, M. J. Australia **1**:631 (May 9) 1936.

Hansman first summarizes the histology of bone, then reviews the extensive literature relating to various phases of calcification.

Osteoblasts are probably present wherever bone is being laid down but do not necessarily arise from embryonic osteogenic tissue, as they have been found in aberrant sites of ossification, such as tuberculous glands.

Osteoclasts are of unproved origin, some saying that they arise by fusion of monocytes; others, that they arise from a single cell by nuclear division. There is a uniform opinion that they are able to cause resorption of the whole structure of bone, but some believe that bone can be resorbed without their aid.

Periosteum at the growing ends of bone forms a ring around the core of diaphysial cartilage and is responsible for the length of the shaft. Failure of the diaphysial mechanism is the cause of achondroplasia.

The matrix of bone has a characteristic microscopic appearance. This appearance can be taken as proof of the presence of bone, but the exact chemical composition of the matrix is as yet unsolved.

Extensive work has been done on bone salts, chemical analysis seeming to indicate that "bone salt is a single substance of the nature of an apatite compound." Investigations from a biologic point of view, however, indicate that calcium carbonate and calcium phosphate are precipitated independently.

The relation of blood to calcification must be studied more completely, as it is becoming evident that serum should be considered not a watery solution but a lyophilic colloid in which, instead of various substances being dissolved in water, the water is dissolved in the protein and the electrolytes exist to a large extent as combinations with the protein. The infrequency of spontaneous calcification and the failure of precipitation of calcium phosphate in serum indicate that the salts do not exist in serum in a state of supersaturation.

The factors influencing the mechanism of calcification are essentially two, an enzyme and a second that favors the deposition of bone salts from supersaturated inorganic solutions. The enzyme is a phosphatase, but there is a large number of phosphatases, and, as calcification takes place only under certain conditions, there must be another factor, with which vitamin D seems to be rather closely linked up. Magnesium plays a peculiar rôle in greatly accelerating phosphatase activity while inhibiting the deposition of bone salt.

The pathologic changes in rickets have been shown to be due to the fact that the second mechanism is most active when the cartilage cells have just obtained their full hypertrophy, so that if vitamin D is given to a rachitic animal the recently hypertrophied cells calcify densely, giving the characteristic line test. Quantitative methods of estimating phosphatase indicate an increase before clinical demonstration of rickets is possible. Also, children showing an increase in phosphatase are kept free from rickets by administration of vitamin D.

The influence of vitamin D in general seems to be indirect, through its action at the site where bone salts are being laid down. There is further evidence that vitamin D does not stimulate the parathyroid glands, but that as far as calcification is concerned vitamin D and the parathyroid hormone may have antagonistic actions. There is some evidence that the importance of vitamin D is a species variant, i. e., it is important for some and unimportant for others.

The relation between the levels of the calcium, phosphorus and vitamin D in the diet has been studied by many. With all elements adequate, if either calcium or phosphorus is reduced, calcification is retarded although normal in character. If only calcium is reduced, the growth of the animal as a whole is unaffected, but the bones show a lower calcium ash. Diets low in phosphorus but otherwise adequate produce normal calcification, but the animals are stunted, showing that reduction in phosphorus has a more serious effect than reduction in calcium. As the animal reaches adult age the bones slowly acquire a normal percentage of ash. In the absence of vitamin D, when the level of either calcium or phosphorus is kept constant while that of the other is raised, rickets becomes intensified.

While most of these experiments have been done on growing animals, changes in the skeleton through deficiency of salt have been proved to occur in adults also.

In contrast to the finding that fetal rickets never occurs in cattle, even when marked deficiency exists in the mother, it has been demonstrated that rickets will occur in the fetus of a mother showing osteomalacia.

Hypervitaminosis D has been demonstrated to cause calcification in tissues, but no definite conclusions can be drawn on this subject as yet.

The effect of endocrine glands on calcification shows that parathyroid hormone above normal requirements causes mobilization of bone salt. In young rats on a normal diet large doses of parathyroid extract caused decalcification, while with moderate doses calcification was definitely increased. In general, conclusions on the effect of parathyroid hormone are that this hormone aids calcification subsequent to primary calcification. In excessive doses it causes mobilization of calcium.

Observations on the effect of the thyroid hormone were inconclusive, although there was some evidence that there may be a hormone in addition to thyroxin.

Thymic extracts have been shown to cause very early maturity in subsequent generations, so that the calcification, while normal, is much accelerated.

Pituitary has been shown to afford a profound stimulus to osteogenesis.

Studies on the excretion and utilization of calcium have shown the requirements to be about 1 Gm. per day, but studies on many persons have shown intakes much below this. The requirements of phosphorus cannot be accurately stated because so much of the phosphorus is needed for metabolic processes other than calcification.

Many problems in relation to calcification are still far from solved, some of which Hansman phrases thus: "What limits the growth and determines the shape of bone? How does the diaphysis know when to join with the epiphysis? How does the bone cell know how much strength is required and when resorption should cease?"

An interesting experiment on new bone formation is reported, showing that when a patella was removed from a puppy a new patella developed only when free activity of the limb was allowed. In other words, the development of the patella was on a functional basis.

TENNEY, Madison, Wis.

THE VALUE OF THE RECTAL ADMINISTRATION OF GLUCOSE. A. B. CORKILL, M. J. Australia **1**:807 (June 13) 1936.

The therapeutic rationale of the administration of dextrose was investigated in an interesting series of experiments. In dogs under amytal anesthesia, which had been previously proved to produce no change in blood sugar, it was found by determinations made on blood from the carotid artery and inferior hemorrhoidal vein that no increase in blood sugar followed injection of dextrose into the rectum, but that after injection of dextrose into the small intestine there was a distinct rise. In studying the effect in man a state of ketosis was induced by means of a ketogenic diet and relieved by immediate change to a diet high in carbohydrate. This was done several times to determine the constancy of the results, as the author had previously found that blood sugar findings in a state of ketosis maintained by a ketogenic diet were inconstant. He induced ketosis by means of ketogenic diet A, but in the relieving diet B he omitted 40 Gm. of dextrose, substituting for it 80 Gm. per rectum, and found that there was no relief from the ketosis. He also tested the relief and prevention of starvation ketosis by dextrose administered by mouth and by rectum, finding again that dextrose given by rectum failed to prevent or relieve ketosis. He concludes that insufficient dextrose is absorbed from the rectum to be of any therapeutic value.

TENNEY, Madison, Wis.

THE MODERN OUTLOOK ON NUTRITION. N. M. GUTTERIDGE, M. J. Australia **1**:835 (June 20) 1936.

The history of nutrition may be divided into six periods: (1) the period of the "heat engine" concept, (2) the vitamin period, (3) the biologic period, (4) the mineral period, (5) the period of balanced composition and (6) the period of action.

Gutteridge says that these terms undoubtedly overlap and regards the first four as the analytic phase and the last two as the synthetic phase. Knowledge of the composition of food is still incomplete but sufficiently far advanced to indicate serious shortcomings in diets in common use. The "heat engine" period carried with it the unfortunate conception of energy in its popular rather than in its physical meaning. Also, the fallacy of adopting the calory as the chief standard of food value is evident from the lack of uniform opinion as to caloric requirements. The vitamin period is likened to the addition of a fourth leg to a chair and the giving that leg credit for supporting the chair, while as a matter of fact the support is equally shared by all. The biologic period is that in which the recognition of so-called protective foods took place, and this really forms the foundation on which the present conception of completely balanced rations is based. The mineral period is represented by the work on iodine deficiency and calcium-phosphorus ratios. The period of balanced composition he regards as the coping stone to the whole nutritional edifice as seen today. The period of action is apparently that of planned correction of glaring deficiencies which have been found in British and Australian dietaries. He also believes that it is going to be necessary to adopt entirely new conceptions of many diseases—for instance, sinus disease, alimentary ulcerations and even cancer of the stomach.

In the light of the glaring deficiencies which have been demonstrated to be present in the normal British and Australian diet entire new standards of what constitutes malnutrition must be adopted.

TENNEY, Madison, Wis.

THE BLOOD KETONE CURVE OF THE CHILD: ITS CHARACTER AND ITS SIGNIFICANCE IN DETERMINING HEPATIC FUNCTION. G. FROLA, Riv. di clin. pediat. **34**:148 (Feb.) 1936.

Although the ketone body content of the blood of both healthy and sick patients during fasting shows certain oscillations, Frola cannot correlate the changes to yield any important information; he does not think that this measurement affords a suitable test of hepatic function or an indication of hepatic insufficiency.

HIGGINS, Boston.

THE INFLUENCE OF INSULIN ON THE BLOOD SUGAR AND BLOOD CALCIUM CURVES OF THE CHILD. G. PAVAN, Riv. di clin. pediat. **34**:481 (June) 1936.

The blood calcium of the child was found to increase following the ingestion of 30 Gm. of dextrose. A similar increase occurred when 3 units of insulin was given at the same time as the dextrose. In rabbits the giving of 1½ units of insulin per kilogram of body weight resulted in a definite increase in the blood calcium. It is apparent that either the production of insulin by the body or its injection into the body leads to an increase in the blood calcium. This action is probably through the vagosympathetic nervous system. HIGGINS, Boston.

THE PHYSIOLOGY OF ANEUTROPHIA. P. DE ALCANTARA, J. de pediat., Rio de Janeiro **3**:253 (June) 1936.

By "aneutrophia" de Alcantara refers to a morbid somatic state in which there is suppression of absorption, assimilation and elimination. This is the third of his articles on the condition, and it further emphasizes this as a distinct condition. Like the other articles, this article is too abstract for a résumé and should be read in full by any one interested. SANFORD, Chicago.

INFANT FEEDING. C. R. CASTILLA, Rev. méd. latino-am. **21**:998 (June) 1936.

In infant feeding it is important to remember that the liver is the organ most susceptible to injury from faulty alimentation. The food should be adapted to the patient, and not standardized. Such factors as race, climate and inherited characteristics should be considered in prescribing a diet.

DWYER, Kansas City, Mo.

IRREVERSIBLE ATHREPSIA. G. MOURIQUAND, Bull. Acad. de méd. de Roumanie **1**:239, 1936.

Mouriquand develops the thesis that at a certain stage in athrepsia irreversible cellular changes occur and "functional death," which resists treatment, sets in. The clinical observations which are cited do not greatly clarify the concept. From the experimental standpoint, work on scorbutic guinea-pigs is considered, and stress is laid on the fact that if treatment is too long delayed in these animals severe gastro-intestinal symptoms supervene, and then treatment is without benefit. The paper contributes little. DIETRICH, Los Angeles.

METABOLISM OF VITAMIN C IN CHILDREN. D. VERHOEFF, Maandschr. v. kindergeneesk. **5**:376 (June) 1936.

Verhoeff concludes from his determinations of vitamin C in specimens of urine (after the method of Tillmans corrected by different authors) from a number of children (partly healthy, partly convalescent) that also in Holland many children are not saturated with vitamin C. Though a small deficiency apparently has no disturbing influence, one may assume that saturation with vitamin C corresponds with the maximal supply of vitamin C. This saturation with vitamin C may be unfavorably influenced by improper feeding, infections and dyspepsia, so that in such conditions it is advisable to take special care that a sufficient amount of vitamin C is given to the child. VAN CREVELD, Amsterdam, Netherlands.

PROBLEM OF SODIUM AND CHLORIDE METABOLISM. P. SCHOORL, Nederl. tijdschr. v. geneesk. **80**:2850 (June 20) 1936.

By means of some quotations from the literature and the argumentation of well known investigators metabolism of salt (NaCl) is denied. Instead of that one has to accept metabolism of sodium completely separated from metabolism of chlorine. VAN CREVELD, Amsterdam, Netherlands.

Vitamins; Avitaminoses

VITAMINIZED FOODS AND COMMERCIAL VITAMIN CONCENTRATES. I. A. MANVILLE, Northwest Med. **35**:209 (June) 1936.

This is a review of the chemical formulas of provitamin A and the vitamins B, G, C, D, E, F and K. The commercial availability of these substances is discussed, with a list of trade names of substances containing them. There is some discussion of the cost of these substances to the public. The biologic value of naturally occurring vitamins is compared with that of isolated and concentrated forms. A very excellent table, containing a list of over fifty preparations, is included.

SCOTT, Philadelphia.

THE TREATMENT OF HUMAN BERIBERI WITH CRYSTALLINE ANTINEURITIC VITAMIN. A. J. HERMANO and FROILAN EUBANAS, Philippine J. Sc. **57**:277 (June) 1935.

The seriousness and extent of beriberi, as well as the types encountered in clinical medicine, are discussed. In twelve cases of beriberi in infants and adults intramuscular injections of crystalline vitamin B_1 gave uniformly good results. Immediate symptomatic relief with ultimate recovery from the disease occurred in practically every case, notwithstanding the fact that the infants, particularly, were admitted to the hospital with severe cardiac and pulmonary symptoms (wet type).

JAHR, Omaha.

PREPARATION OF A CONCENTRATON OF VITAMINS B_1 AND B_2 FROM BREWERS' YEAST. MAURICE I. SMITH and ATHERTON SEIDELL, Pub. Health. Rep. **51**:685 (May 29) 1936.

Smith and Seidell prepare the concentrate by precipitating the extracts with alcohol and fullers' earth and with vacuum distillation. Their unit for B_1 is the international standard unit; for B_2, a preparation 200 mg. of which produces a supraminimal increment of weight of about 1.5 Gm. per day when fed over a period of ten days.

The international standard for B_1 is not wholly free from B_2, though it is only about one third as active as dried brewers' yeast. Concentrates prepared by the authors' method have a B_1 potency of from sixty to five hundred times that of brewers' yeast and a B_2 potency of from seventy to one hundred and seventy-five times. In experimental dermatitis in rats, from 200 to 300 mg. of dried brewers' yeast cures the condition in from ten to twenty days, while 10 mg. of concentrate assaying 500 units per gram, when given intravenously, cures in six days.

SANFORD, Chicago.

KWASHIORKOR. CICELY D. WILLIAMS, Lancet **2**:1151 (Nov. 16) 1935.

Kwashiorkor is the local name in the Gold Coast for a nutritional disease of children which usually occurs between the ages of 6 months and 4 years, and is associated with a maize diet. The victims show no evidence of scurvy or rickets. It has been suggested that it may be a form of pellagra, but it seems to differ in a number of respects from this disease.

LANGMANN, New York.

THE BIOCHEMICAL LESION IN VITAMIN B_1 DEFICIENCY: APPLICATION OF MODERN BIOCHEMICAL ANALYSIS IN ITS DIAGNOSIS. R. A. PETERS, Lancet **1**:1161 (May 23) 1936.

New methods of biochemical analysis of enzymes were applied by workers in the department of biochemistry of Oxford University to the problem of the origin of symptoms of acute opisthotonos in pigeons suffering from a deficiency of vitamin B. A defect in the power of oxidizing certain carbohydrate intermediates in the

central nervous system, chiefly lactic acid ($CH_3.CHOH.COOH$) and pyruvic acid, was found. Minute amounts of crystalline vitamin B added to the avitaminous brain tissue in vitro restored the diminished respiration of tissue to normal.

LANGMANN, New York.

INFANTILE SCURVY IN CHILE. E. CIENFUEGOS, Arch. de pediat., Rio de Janeiro **7**:414 (June) 1936.

Ten cases are reported. The youngest patient was 7 months of age. Six of the patients were girls, and four, boys. The etiologic factor was the consumption of condensed milk with no accessory antiscorbutic substance. All the patients had painful joints; seven had gingival manifestations; one had hematemesis, and one, subcutaneous hemorrhage.

SANFORD, Chicago.

RELATION OF HYPOVITAMINOSIS TO DARK ADAPTATION. M. NAGY and K. INCZE, Arch. f. Augenh. **109**:567, 1936.

In 1934 Jeans and Zentmire (Clinical Method for Determining Moderate Degrees of Vitamin A Deficiency, *J. A. M. A.* **102**:892 [March 24] 1934) reported observations on a series of children who were known to be on a vitamin-poor diet and who showed a deficiency in dark adaptation. This was the sole evidence of hypovitaminosis, and the dark adaptation returned to normal when additional vitamin A was added to the diet.

The present authors examined a series of children in two different schools who were known to be on diets deficient in vitamins. The children in a third school, whose supply of vitamins was adequate, were used as a control. The dark adaptation of the children in these two groups was measured by means of the five point adaptometer of Birch-Hirschfeld, which gives measurements similar to those taken by Jeans and Zentmire.

Nagy and Incze summarize their results as follows: 1. No differences in dark adaptation could be found in children who had diets varying widely in vitamin content. 2. In the spring of the year, when the vitamin content of the body was definitely low, there was a diminution in the power of dark adaptation, and in the fall this impairment had disappeared. The examinations were carried out first in the fall and then in the spring, so that the better power of adaptation on the part of the children in the fall would not be due to experience with the test.

ADLER, New York. [ARCH. OPHTH.]

HEMORRHAGE AND VITAMIN C. GEORG OSKAR HARNAPP, Monatschr. f. Kinderh. **66**:308 (Aug.) 1936.

Harnapp treated various types of hemorrhagic disease with large doses of cevitamic acid intravenously. He concludes that the treatment was of no practical value.

GERSTLEY, Chicago.

RICKETS. E. MÜLLER, München. med. Wchnschr. **83**:855 (May 22) 1936.

Müller reviews the history of rickets from its discovery in England in the early seventeenth century and the description by Glisson to the discovery of irradiated ergosterol by Windaus, Pohl and Hess. The article is interesting but does not lend itself to abstracting.

BRAHDY, Mount Vernon, N. Y.

NEW SOURCES OF VITAMIN A AND THEIR USES. PETER G. MAR, Chinese M. J. **50**:1086 (Aug.) 1936.

Mar, working in the division of physiologic sciences of the Henry Lester Institute of Medical Research, Shanghai, reports a recent study of some potential sources of vitamin A and provitamin A, being guided in selecting the material by remedies recommended in the ancient medical literature of China for the treatment of night blindness. He quotes Professor Read, who in 1934 published

a paper "The Newer Pharmacology and Its Relationship to Ancient Medicine," in which it was shown that there were many references in ancient medical records to the deficiency diseases and their treatment.

It seems logical to consider that in China the origin of night blindness is nutritional. Poverty and unequal distribution of food due to many causes subject the middle and lower classes to nutritional deficiency. The author lists legumes, cereals, vegetables, fruit and egg as Chinese foods which were used in estimating the content of vitamin A. One table deals entirely with the potency of the carotenoids. There is listed also the provitamin A content of foods and drugs, especially the vegetable remedies for night blindness.

NEFF, Kansas City, Mo.

THE RACHITOGENIC FACTORS IN CEREALS. O. RYGH, Norsk. mag. f. lægevidensk. **97**:401, 1936.

The free fatty acids of oatmeal were found to contain rickets-producing factors. This rachitogenic effect was measured against the antirachitic effect of the fatty acids of the green vegetables.

TOVERUD, Oslo, Norway.

Hygiene; Growth and Nutrition; Public Health

DEPRESSION CHILDREN. I. N. KUGELMASS, Arch. Pediat. **53**:386 (June) 1936.

Notwithstanding the lowering of the infant morbidity and mortality rates during the years from 1927 to 1932, Kugelmass is under the impression that the depression has had an intangible but devastating effect on the mental and physical health of the nation's children.

ORR, Buffalo.

CURRENT PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES. Pub. Health Rep. **51**:731 (June 5) 1936.

For the period of April 19 to May 6, 1936, there was a decline in influenza, meningococcic meningitis, scarlet fever, typhoid, diphtheria and measles, the incidence of which was only about 40 per cent of that of the last two years. There were 956 cases of smallpox, which is the highest incidence of the last four years. There were 64 cases of poliomyelitis, an increase of 35 per cent over the preceding four week period, but the current incidence is the lowest in the last eight years. The current death rate was 12.7 per thousand, the highest for the period since 1929.

SANFORD, Chicago.

THE RELATION OF PHYSICAL DEFECTS TO THE PHYSICAL GROWTH OF CHILDREN OF TWENTY-ONE STATES. WILLIAM M. GAFAFER, Pub. Health Rep. **51**:831 (June 26) 1936.

The measurements taken were those of weight, standing height, sitting height, circumference of the chest, transverse diameter, anteroposterior diameter and vital capacity. The material was 30,000 elementary school children of native-born white parents in twenty-one states. The children of the nondefective group were on the average taller and heavier and had longer trunks and greater vital capacity. They were stockier, and in relation to height they had short trunks and small chest girths. The groups showed no difference in rates of growth.

SANFORD, Chicago.

THE INFLUENCE OF RAW AND STERILIZED MILK ON GROWTH AND REPRODUCTION IN RATS. H. A. CHANNON and H. J. CHANNON, J. Hyg. **36**:173 (June) 1936.

Milk homogenized and sterilized at 212 F. for thirty minutes was fed, plus white flour biscuits, iron, copper and manganese, to litters of rats for periods up to one year.

As compared with a control group given the same milk raw, the group given sterilized milk showed weights 10 per cent less.

Both groups reproduced but failed to rear their litters.

JANNEY, Wauwatosa, Wis.

EDUCATION IN OPEN AIR SCHOOLS IN ENGLAND AND WALES. JAMES LUMSDEN, Ztschr. f. internat. Kom. f. Freiluftschulen, 1936, p. 7.

This article has to do with the principles which guide the teacher in drawing up schemes of work for their pupils in the open air schools. Since most of the children are backward through illnesses and consequent frequent absences, no effort is made to push them in their studies.

The time table of the school is so flexible that no formality of keeping to the hour and minute should deprive the children of the opportunity of observing nature, which may not recur when they return to their more sordid surroundings. The instruction should be suited to the capacity of dull as well as to that of normal children, and the classes should remain small so that individual needs may be met. Each lesson should be designed to interest the children so that they will work keenly without strain. The whole environment leads to a more practical type of education than can be worked out in the classroom of a crowded city school. It is hoped that these children will return to their city schools renewed in health and strength and with a renewed interest in learning.

LESLIE, Evanston, Ill.

Prematurity and Congenital Deformities

ECTOPIA CORDIS CUM STERNI FISSURA. C. E. KELLETT, Arch. Dis. Childhood **11**:205 (Aug.) 1936.

A case of ectopia cordis, with the observations at autopsy, is described. Electrocardiograms and electrographs were taken, and the effects of cold on the electrographs were recorded. It is felt that the interpretation of these results is dependent on a further experimental investigation, but by virtue of the rarity of the conditions under which they were taken they appear to merit recording.

KELLY, Milwaukee.

PERSISTENT OMPHALOMESENTERIC DUCT. B. SANCHEZ SANTIAGO, Arch. de med. inf. **5**:275 (Aug.) 1936.

A child with a persistent omphalomesenteric duct also had congenital heart disease. The duct may show a complete, partial or incomplete fistula, with occlusion toward the intestines, while it is freely suspended from the navel, or it may be fastened to the intestines with a fibrous cord. The malformation produces distress when the bowel is emptied and may at any time tear the intestine, producing strangulation. The open diverticulum should be removed surgically, although the life of the child is in no immediate danger. SANFORD, Chicago.

FACIOCRANIAL DYSOSTOSIS (CROUZON'S DISEASE) IN A HEALTHY FAMILY. P. BURTON and AUGUST O. LEY, J. belge de neurol. et de psychiat. **35**:57 (Feb.) 1935.

Burton and Ley present the case of a patient with Crouzon's disease, the important features of which they describe as (1) cranial deformity similar to oxycephalia; (2) malformation of the face with flattening of the nose and superior micrognathia giving way to inferior prognathia, and (3) ocular difficulties, such as atrophy of the optic nerve, exophthalmos and divergent strabismus. Although the syndrome is considered familial and hereditary, it may be seen as an isolated condition. The case reported is that of a child aged 3½ years, who at the age of 3 months had hydrocephalus with exophthalmos and strabismus but no atrophy of the optic nerve. There was at that time a fairly typical facial expression. The

family and developmental history is without significance except for delay in walking. A roentgenogram showed the bones of the skull to be thin, with symmetrical deformities on both sides, visible fontanels, a rather opaque cerebral vault, small maxillary sinuses and normal dentition. The vestibular apparatus was not stimulated by caloric tests. A lumbar puncture revealed high pressure. The fluid was normal. At the age of 3½ years the intelligence quotient was rated as between 18 months and 2 years.

WAGGONER, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

DYSCEPHALODACTYLISM (VOGT) AND DEVELOPMENTAL ANOMALIES OF THE UVEAL TRACT. K. INCZE, Arch. f. Augenh. **109**:562, 1936.

Two cases are reported of developmental anomalies of the skull and of the uveal tract which seemed to show a hereditary tendency. The first case was that of a 42 year old man who had in one iris a brown cyst of spontaneous origin, which disappeared after an x-ray picture was taken of the skull. Incze rightly points out that this disappearance was merely a coincidence. Associated with the changes in the skull were deformities in the lower extremities, i. e., syndactylism.

The second case was that of a 7 year old boy with coloboma of the iris, epicanthus and deformities of the skull. ADLER, New York. [ARCH. OPHTH.]

HEREDITY, CONSTITUTION AND INJURY TO THE GERM PLASM. HANS LUXENBURGER, Fortschr. d. Neurol., Psychiat. **7**:173 (May) 1935.

This article is a review of the major contributions in the fields indicated in the title.

The work of Schulz on the heredopathology of schizophrenia, Luxenburger's work on cyclothymia and Abadie's on epilepsy seem to be the most outstanding contributions of the year. In substance, Schulz' report is based on the idea that nothing happens accidentally and that everything in genetics follows the laws of causality.

Abadie denied the heredity of epilepsy; he was also doubtful as to the conception of the so-called epileptic constitution. Instead of dividing epilepsy into "genuine" and "symptomatic," Abadie divided it according to age. The juvenile forms belong to the juvenile eclampsias. The adult forms are divided into traumatic, puerperal, syphilitic, malarial, pleural and cardiac and the epilepsy of pregnancy (eclamptic). It is not alcoholism of the parents which gives rise to epilepsy in from 30 to 70 per cent of the descendants but rather the incidental birth injuries and accompanying infections.

Gebbing in his "Die Erbanlage bei Neurotikern" investigated 100 family trees consisting of 466 persons in order to study the hereditary predispositions of the neurotic persons. Of 340 living persons, only 201 could be studied individually. It was found that there is a familial predisposition to neuroses. A relationship between neurosis and vegetative lability could not be established.

Sjoegren systematically studied mental deficiency in a rural settlement in the Northwest. In this group he found an oligophreniform type which appeared 52 times in 34 families. There was a high percentage of intermarriage among them with a high incidence of hereditary feeblemindedness. The hereditary taint is recessive and monomeric. Sjoegren expressed the belief that the disease is determined partly by autosomes and partly by sex-attached genes. Fifty-three per cent of the mothers of this group were blood relatives; only 14 per cent of the fathers were related. The "anlage" seems to pass more easily through the mother than through the father.

Delfini, discussing amniogenic idiocy, concluded that syphilis is an essential etiologic factor. Lang in a study of cretinism discussed the question of radio-activity in relationship to goiter. He studied the medical, geologic and physical aspects and concluded that (*a*) with the increase of radio-activity there is an increase in the number of goiters and that (*b*) this parallelism pertains to the total radio-activity as well as to isolated alpha and gamma rays.

Neippberg investigated thoroughly color blindness in a family tree which had 1,189 descendants in 7 generations. Of 578 males, 39 were color blind. Of 611 females, only 4 were color blind.

Achard, Betrand and Escalier described the case of a woman, aged 30, who had cerebellar ataxia, spastic paraplegia with parkinsonism and psychic disturbances. Autopsy revealed lesions in the spinocerebellar and pyramidal tracts. On the maternal side there were 7 diseased persons in 3 generations. Most of them had symptoms similar to those manifested by the patient. The patient first became ill after a pregnancy at the age of 24 and then again at 37. At the time of the investigation she was 44. The chief symptoms were: mild exhaustibility, dizziness, unsteady gait, staccato speech, ptosis, Argyll Robertson pupils, nystagmus, paresis of the seventh and twelfth cranial nerves, dysphagia, tremor of both hands and cerebellar ataxia of the limbs. The mother of the patient, aged 63, became ill at 30 after an attack of typhoid. Three children of the patient show the disease in abortive forms. The maternal grandmother suffered from dysphagia and dysarthria. The disease appeared rather late in all members of the family. The ocular symptoms and dysphagia were predominant. Variation in the clinical picture is due, according to these authors, to trauma and infection. They also assume that there is a genetypic variability because of the process of mutation, which may have differed in different instances. One should then accept that there are numerous independent but closely similar genes. The unmistakable intrafamilial similarity of the clinical picture points in this direction. Roussy and Levy reported on a family in which there were 20 cases of hereditary disturbance of gait complicated by areflexia.

Barnes described many cases of myopathy in a large family in several generations. From a diseased father there were 500 descendants in seven generations. Of the 284 investigated descendants, 41 were diseased; the disease was distributed among five generations. Those affected presented a characteristic picture of pseudohypertrophy with progressive muscular weakness and atrophy of the lower and hypertrophy of the upper extremities. The difference in this type of atrophy from the Charcot-Marie-Tooth type is seen in the predilection for the proximal muscles, especially the iliopsoas muscles, and the absence of sensory and reflex disturbances. Barnes expressed the belief that this condition is a disease in itself. The onset of the disease is in middle age, between 35 and 50. The course is chronic but may be hastened by trauma or infection. The disease is genetically dominant.

Hippel-Lindau's disease with angiomatosis of the retina and capillary angiomatosis of the central nervous system is a familial disease and is dominant. Often it is not recognized because of deficient phenotypic signs. In Kufs' case there was capillary angiomatosis of the cerebellum with cystic formations in a man aged 30. An injury to the head preceded the onset of the trouble. No other cases were found in the family.

Of considerable importance is the work of Iljon dealing with epilepsy and chronic hyperkinesis in 5 brothers; 1 sister was normal. Two other siblings died during childhood. Hyperkinesis first appeared at 11, becoming chronic. After two or three years nocturnal epileptic attacks occurred. Finally, ataxia, hypotonia and dementia ensued. ALTSHULER, Detroit. [ARCH. NEUROL. & PSYCHIAT.]

INVESTIGATIONS ON IDENTICAL TWINS WITH RESPECT TO RICKETS AND TUBERCULOUS ADENITIS. L. J. BOSSIK and E. I. PASSYNKOFF, Monatschr. f. Kinderh. **66**:352 (Aug.) 1936.

The writers studied the effects of irradiation with the mercury vapor lamp in sixteen cases of rickets and twenty-three of tuberculous bronchadenitis. Each patient was one of a pair of identical twins, the sibling being used as a control. The treatment was of definite value in each of these diseases. The beneficial effect of the ultraviolet rays remained manifest for about six months in the cases of rickets and about ten months in the cases of adenitis. There was no protection afforded against other types of infections, such as grip and colds, by the rays. The beneficial effect of the ultraviolet rays was independent of the dose.

GERSTLEY, Chicago.

FAMILIAL FLEXION CONTRACTION OF THE FINGERS. P. RITTERSKAMP, München. med. Wchnschr. **83**:725 (May 1) 1936.

Ritterskamp describes a flexion contraction of the first interphalangeal joint of the little finger in a 60 year old man. The condition had occurred in five other members of the family in the last three generations. Two of these were examined. One was a 24 year old girl and the other a 50 year old woman, in both of whom the contracture was symmetrical. The condition usually involves the little finger but has been described in the third and fourth fingers.

BRAHDY, Mount Vernon, N. Y.

THE ROENTGENOGRAPHIC VISUALIZATION OF A COMPLETE LATERAL CONGENITAL FISTULA OF THE NECK WITH IODIZED POPPY-SEED OIL. H. GOLLUB, Röntgenpraxis **8**:388 (June) 1936.

A 20 per cent solution of iodized poppy-seed oil provided a satisfactory contrast medium for the purpose stated in the title.

McCUNE, New York.

THE PREPARATION OF TEACHERS FOR THE OPEN AIR SCHOOL. M. J. FORCEVILLE, Ztschr. f. internat. Kom. f. Freiluftschulen, 1936, p. 2.

In this brief article Forceville reviews the qualifications necessary for the teacher in the open air school. Physical strength and endurance are of the utmost importance, as the task here is harder than that in the ordinary school. Also, suppleness of intellect and vivacity of spirit combined with a well balanced nervous system are necessary in order to achieve the best results with the handicapped pupils.

LESLIE, Evanston, Ill.

New-Born

EXTRADURAL HEMORRHAGE AS A RESULT OF BIRTH TRAUMA. L. L. LEFKOWITZ, Arch. Pediat. **53**:404 (June) 1936.

Lefkowitz reports a case of epidural hemorrhage produced by a trauma sustained at birth. The infant was delivered by version and extraction and lived for six days. The autopsy showed considerable overriding of the cranial bones but no fractures. An epidural clot was found in the right temporal region.

ORR, Buffalo.

PREVENTION OF LOSS OF WEIGHT IN THE NEWBORN. J. I. WARING, J. South Carolina M. A. **32**:188 (Aug.) 1936.

The possibility of preventing the initial and the normal physiologic loss of weight in the new-born baby is dubious. The best solution to use is lactose or lactose citrate, and not a milk mixture, as the latter might interfere with breast feeding and not infrequently causes a sensitivity reaction.

OBERTON JR., Columbia, S. C.

CONGENITAL PEMPHIGUS NEONATORUM. WILLIAM WESTON JR., South. M. J. **29**:905, 1936.

Weston reports a case of congenital pemphigus which involved the entire skin (except the scalp), the nails and the mucous membranes of the mouth and throat. The etiology in this case is unknown, but there is a suggestion of an infectious origin. No treatment seemed to be effective.

SCHLUTZ, Chicago.

NASAL OBSTRUCTION IN THE NEWBORN. A. MONCRIEFF, Brit. M. J. **1**:1295 (June 27) 1936.

This is a very timely article on various kinds of congenital obstruction of the nasal passages, with a comment on these as a possible cause of death. Moncrieff emphasizes the desirability of investigating the nasal passages of any child who does not breathe well, especially when nursing.

ROYSTER, University, Va.

NEO-NATAL MORTALITY IN DUBLIN. W. R. F. COLLIS, Irish J. M. Sc., May 1936, p. 193.

The first factor discussed is the temperature. Thyroid extract is used in cases in which the temperature is lower. All premature and weak babies are given 0.25 grain (0.016 Gm.) of thyroid and then 0.1 grain (0.006 Gm.) twice daily for from three to four weeks. The nursing technic in handling premature infants is stressed. The question of respiratory infection is also stressed since many premature and weak babies are lost through this cause. In cases of trauma sustained at birth the babies are given tenth-normal saline solution rectally to reduce the cerebral edema. Later lumbar puncture is done to relieve the intracranial pressure. With the institution of these measures as well as the treating of anemia, atelectasis, congenital syphilis and the other neonatal diseases, the mortality in premature and weak babies has been decreased from 50 to 20 per cent.

SNELLING, Toronto, Canada.

CASE OF BILATERAL RENAL INFARCTION IN A NEWLY BORN INFANT. STANLEY MARSHALL and EILEEN WHAPHAM, Lancet **2**:428 (Aug. 22) 1936.

The case of a boy whose birth appeared perfectly normal and who weighed 9 pounds and 2 oz. (4,139 Gm.) at birth is reported. The placenta was normal. On the day after birth he took the breast reluctantly and voided but a scant amount of urine. He was weaned because of the poor secretion of breast milk. A little blood-stained urine was noticed on the ninth day. On the thirteenth day he was circumcised. The following day there was bleeding from the umbilicus. Vomiting set in, and he died on the twentieth day.

Postmortem examination revealed a rather large heart, with small hemorrhagic blebs along the free edges of the mitral and tricuspid valves. The kidneys presented a remarkable appearance. The two together occupied almost a third of the total abdominal space. Section showed a smooth reddish brown surface with the cortex and medulla in normal ratio. The whole kidneys were apparently tightly distended by a diffuse hemorrhage into their substance. On histologic examination large areas of necrosis were seen to alternate with areas of fibrosis and hemorrhage. The impression gained was that of a recent infarction involving almost the whole kidney substance, superadded to some previous change, presumably of a similar nature, which must have occurred in utero.

Various possibilities were considered, such as maternal disease, acute nephritis, hemorrhagic disease and Winckel's disease, but no light was thrown on the etiology.

LANGMANN, New York.

SCOLIOSIS OF THE NEW-BORN. R. J. HARRENSTEIN, Rev. d'orthop. **23**:289 (July-Aug.) 1936.

Harrenstein limits his discussion to scoliosis without skeletal changes, occurring in infants between 3 and 6 months of age. There may or may not be alterations in the contour of the chest. This condition has received little prominence in the literature, and the etiology appears still obscure. In the infants studied the scoliosis was not apparent at birth but developed about the third to the sixth month. Therefore intra-uterine malposition could not have had any bearing on the development of the condition. Neither rickets nor the position in which the mother carried the child seemed to have had any bearing on the disorder. The

calcium content and the phosphorus content of the blood were within normal limits. Whether muscular imbalance produced the condition has been hard to prove, although in cases in which such imbalance has been notably present scoliosis has accompanied it.

Treatment has been by the use of plaster half-shells and corsets. The author has devised an oak board much like a punch-board in appearance, on which the child is laid face downward. Iron pegs are placed in this board in such a way as to hold the child in the corrected position. Cotton padding is placed over the points of pressure, and the plaster half-shell is constructed with the child held in the corrected position. The child is kept in the dorsal position in the half-shell for twenty hours each day. During the remaining four hours the child is kept face downward in a corset. This lessens the liability of atrophy of the musculature of the back. As improvement occurs the period in which the child is kept in the dorsal posture is lessened and the period in which he is kept in the ventral posture increased. Treatment is continued over a period of from one to two years. The results have been good. Approximately half of the children so treated have remained without further deformity. One difficulty in the treatment is obtaining the cooperation of the parents.

RYAN, Vancouver, Canada.

CRANIAL DEPRESSION IN THE NEW-BORN. J. M. BERNAT, Rev. méd. latino-am. **31**:923 (May) 1936.

Cranial depressions in the new-born are not as common as formerly, owing to better deliveries and more prenatal care. Bernat believes that depressed bones of the skull should never be left untreated regardless of the absence of symptoms. The best method of correcting such injuries is through the sutures. Removal of pieces of bone should be left for complicated cases. Six cases are reported, in which cranial depressions were reduced with good results.

DWYER, Kansas City, Mo.

INJURIES SUSTAINED AT BIRTH: THEIR PREVENTION, DIAGNOSIS, TREATMENT AND PROGNOSIS, H. NAUJOKS, München. med. Wchnschr. **83**:835 (May 22) 1936.

The prevention of injuries at birth is dependent on careful obstetrics. Whether operative intervention in a delivery will be necessary should be determined early and not when an emergency has arisen. The most frequent mistakes in the application of forceps are (1) application of pressure too suddenly or too long, (2) rotation of the blade and (3) slipping of the blade from the head. It should always be borne in mind that the forceps is not for the application of pressure but for traction. It should not be used to compress a large head for a small pelvis. Traction should be applied slowly and rhythmically in conjunction with the birth pains. High forceps should not be applied by the general practitioner. Among other detailed instructions, the obstetrician is warned against torsion of the extremities which may cause epiphysial separation or injury of a joint.

The new-born infant should have a thorough physical examination. A roentgenogram is often necessary to determine the presence of certain fractures and injuries of joints. Symptoms of intracranial injury may not become manifest for several days.

Fractures, injuries of the brachial plexus, dislocated joints and trauma of the sternocleidomastoid muscle are treated by appropriate immobilization, in casts if necessary. If there is respiratory difficulty after the air passages have been cleared in an infant with intracranial injury, he should be given lobeline, coramine (a 25 per cent solution of pyridine betacarbonic acid diethylamide) and carbon dioxide to stimulate the respiratory center. The body should be kept warm and the head cool. The coagulability of the blood may be enhanced by injections of calcium salts, blood serum or gelatin. Convulsions should be controlled with chloral hydrate and magnesium sulfate. The infant's nutrition is of great importance, and gavage or rectal feeding may be necessary.

Naujoks, an obstetrician, does not share the pessimistic attitude of pathologists and pediatricians concerning the ultimate fate of infants with intracranial injuries sustained at birth. The fact that a child who was delivered with forceps later has Little's disease or becomes an imbecile does not necessarily prove a causal relation. The author has not seen Little's disease develop in any of his patients who had intracranial hemorrhage at birth. He believes that heredity has not been stressed sufficiently in explaining cerebral conditions occurring in childhood. The parents of a child with a cerebral hemorrhage can be told that if he withstands the first attacks his chances of normal development are good.

BRAHDY, Mount Vernon, N. Y.

COMPARISON OF THE O SUBSTANCE IN NEW-BORN INFANTS AND IN ADULTS. B. ELMENHOFF-NIELSEN, Ztschr. f. Rassenphysiol. **8**:81, 1936.

By means of titration and absorption tests with the α_2 agglutinins present in certain ox serums the author found that the O receptor in group O new-born infants had reached a fourth of the usual strength. On the other hand, the O substance was found to be present in larger amounts in new-born infants of groups A and AB than in adults of groups A and AB. This was true especially of the new-born infants in whom the A receptor was least developed. By immunizing a rabbit possessing preformed α_2 agglutinin with group O blood, an immune anti-O serum was obtained, though not of a very high titer (approximately 1:256).

WIENER, Brooklyn. [ARCH. PATH.]

FIXATION OF THE HEAD OF THE FETUS DURING PREGNANCY. H. MARCUS, Acta obst. et gynec. Scandinav. **16**:356, 1936.

In 5,422 observations of 184 primiparous and 301 multiparous pregnant women the head of the fetus was found movable in three-fourths of the observations. The proportion of such observations in primiparas is slightly smaller than that in multiparas.

In a third of the cases, divided equally between the two groups, alternations between fixation and movability took place up to delivery. The factors which influence fixation or movability are the absolute and relative size of the fetus in relation to the pelvis, the effect of fetal movements, the amount of amniotic fluid, the condition of the lower uterine segment, the effect of the contractions of the uterus and the nature of the abdominal wall.

ADAIR and POTTER, Chicago.

INJURIES OF THE CENTRAL NERVOUS SYSTEM SUSTAINED AT BIRTH. ERIK RYDBERG, Hospitalstid. **79**:453 (May 5) 1936.

Rydberg reports on the ultimate effects of intracranial injuries sustained at birth in forty-eight children from three to eighteen years after birth: idiocy or imbecility in sixteen; psychic debility in ten; severe somatic defects but with intelligence normal in two; jacksonian fits in one; defects of less importance in ten. Nine of the children were normal. His prognosis in such cases is as follows: A third of the patients have residues so severe that they are dependent on the help of other people; a third can take care of themselves, and a third show severe injuries of uncertain prognosis.

WALLGREN, Göteborg, Sweden.

SPONTANEOUS RESTORATION OF THE NORMAL SHAPE OF THE FEMUR AFTER HEALING OF A FRACTURE IN THE POSITION OF FLEXION: A CASE IN A NEW-BORN INFANT. T. A. JAGERINK, Maandschr. v. kindergeneesk. **5**:344 (May) 1936.

A fracture of the upper part of the femur in a new-born infant healed with marked angulation. After three years the angulation has nearly disappeared, and the femur has practically gained its normal form and function.

VAN CREVELD, Amsterdam, Netherlands.

HIGH DESTRUCTION OF THE BLOOD IN THE NEW-BORN. J. H. P. JONXIS, Maandschr. v. kindergeneesk. **5**:363 (June) 1936.

The high percentage of hemoglobin in the blood of the new-born is generally considered to indicate an adaptation of the fetus to the imperfect supply of oxygen during pregnancy. Starting from the well known researches of Barcroft and those of Hasselhorst and Stromberger, Jonxis discusses the low oxygenation of the fetal blood. The oxygen supply of the fetus varies in the different parts of the body through the mixing of arterial and venous blood. The liver and the upper part of the body are best supplied with blood, whereas the lower part grows almost without oxygenated blood. The causes of the insufficient oxygenation of human fetal blood are mentioned.

The conception that the high percentage of hemoglobin in the fetal blood represents an adaptation to the insufficient supply of oxygen is rejected, also the theory of Anselmino and Hoffmann as to the cause of icterus neonatorum, which is based on this conception. The cause of the destruction of hemoglobin in the new-born is not a better supply of oxygen after birth but the disappearance of fetal, and the appearance of later, hemoglobin.

VAN CREVELD, Amsterdam, Netherlands.

THE POSSIBILITY OF THE CHILD'S BEING ALIVE AT BIRTH WHEN THE UMBILICAL CORD IS NONPULSATING. S. A. SCHOUSTRA, Nederl. tijdschr. v. geneesk. **80**:33 (Jan. 4) 1936.

A case of parturition is described in which a nonpulsating cord was found and the child was delivered by forceps after a vain attempt at version and extraction. The time which elapsed between the incarceration of the cord and the delivery of the asphyctic child was probably not less than ten minutes.

VAN CREVELD, Amsterdam, Netherlands.

Acute Contagious Diseases

THE VISCERAL PATHOLOGY IN SCARLET FEVER AND RELATED STREPTOCOCCUS INFECTIONS. H. BRODY and L. W. SMITH, Am. J. Path. **12**:373 (May) 1936.

A study of visceral lesions in scarlet fever and related streptococcic infections is presented. Material was examined from forty-four cases of frank scarlet fever and fifteen cases of presumptive scarlet fever. The literature covering the last seventy-five years is reviewed in some detail. The studies reveal that the underlying lesion is a vascular injury with concurrent perivascular round cell infiltration. These changes have been found in from 75 to 95 per cent of the hearts, livers, kidneys, adrenal glands and spleens and to a variable degree in the other viscera, including the pituitary gland, lung, pancreas and even the testis. The evidence suggests that the primary lesion results from the action of a circulating toxin. This is borne out by a certain amount of experimental evidence, which is reviewed briefly.

FROM THE AUTHORS' SUMMARY.

THE HISTOLOGY OF EXPERIMENTAL MUMPS IN THE MONKEY. C. D. JOHNSON and E. W. GOODPASTURE, Am. J. Path. **12**:495, 1936.

In experimental mumps the essential lesion is the result of a focal action of the mumps virus on acinar epithelial cells, resulting in degeneration and necrosis of these cells. The inflammatory cellular response is secondary to the specific injury and consists of an infiltration of the area by mononuclear phagocytic cells and later by various types of lymphocytes. Polymorphonuclear leukocytes play no part in the reaction. Dissemination of lesions within the infected gland

follows. The edema is probably dependent on a diffuse vascular injury secondary to the focal parenchymal lesions. Healing takes place by removal of the débris and regeneration of the acinar epithelium, which restores the gland without scarring. Inclusions in the affected epithelial cells are described and are interpreted as specific. It is concluded that in all probability similar lesions underlie mumps in human beings. FROM THE AUTHORS' SUMMARY. [ARCH. PATH.]

WHOOPING COUGH IN TUBERCULOUS CHILDREN. J. IVAN HERSHEY and FRIEDA WARD, Am. Rev. Tuberc. **32**:612 (Nov.) 1935.

A small series of cases of whooping cough occurred among tuberculous children in a sanatorium. In each case confirmatory diagnosis was established by cultures on cough plates. A vaccine prepared from the strain of Bacillus pertussis isolated from these patients was employed in both the prophylactic and the active treatment. Clinically, none of the children exhibited any ill effects from the whooping cough; in fact, most of them benefited greatly from the enforced rest in bed. Roentgenographically, six of the nine children showed only a slight generalized accentuation of the trunk markings of the lungs, while in the other three instances there were no changes whatsoever. This is in marked contrast to the generally accepted view that whooping cough is a grave complication in cases of latent or active pulmonary tuberculosis.

EUGENE SMITH, Ogden, Utah.

REPORT OF A CASE OF SEVERE WHOOPING COUGH RESPONDING TO PERTUSSIS VACCINE (SAUER). FREDERICK STENN, Illinois M. J. **68**:86 (July) 1935.

Stenn reports a case of whooping cough in which the patient apparently benefited by the administration of pertussis vaccine (Sauer).

BARBOUR, Peoria, Ill.

CONTRIBUTION TO WHOOPING COUGH IMMUNIZATION. CARL E. SIBILSKY, Illinois M. J. **68**:549 (Dec.) 1935.

Sibilsky gave seventy-five children injections of pertussis vaccine (Sauer) for immunization. Six of the children were later intimately exposed. Whether the others were exposed or not is not known. None of the children who were given the vaccine contracted whooping cough.

BARBOUR, Peoria, Ill.

FACTORS OF RESISTANCE IN EXPERIMENTAL POLIOMYELITIS. N. P. HUDSON, E. H. LENNETTE and F. B. GORDON, J. A. M. A. **106**:2037 (June 13) 1936.

Studies were made regarding the resistance of the experimental animal Macaca mulatta to acute anterior poliomyelitis. Although ample evidence pointed to the upper respiratory tract as the portal of entry of the virus, a certain degree of resistance was demonstrated in the nasopharyngeal mucosa. The intestinal mucosa was an effective barrier when the virus was administered to isolated intestinal loops. Splenectomy seemed to reduce the resistance in two of eleven animals when the operation was performed before injections of the virus. The virus was found in the spleen in the first twenty-four hours after intravenous or intrasplenic injection. Neutralizing antibodies were formed in monkeys vaccinated with certain preparations, but this was no indication of protection against intranasally introduced virus. Menstruation did not produce a demonstrable virucidal property of the blood. Selectivity of the olfactory tracts for the virus was demonstrated by the fact that section of these tracts prevented infection by the intranasal and intravenous routes. Further selectivity was shown by recovery of the virus from the nasopharynx in monkeys infected by way of the blood stream. These experiments mean that poliomyelitis is primarily and largely of the nervous system.

BONAR, Salt Lake City.

EXPERIMENTAL MENINGOCOCCAL INFECTION IN THE MOUSE. C. P. MILLER and R. CASTLES, J. Infect. Dis. **58**:263 (May-June) 1936.

"It was possible to initiate fatal infections regularly in mice by the intraperitoneal inoculation of very few meningococci suspended in mucin prepared from hog's gastric mucosa. With our most virulent strains approximately ten organisms sufficed.

"Multiplication of meningococci inoculated into the peritoneal cavity in mucin suspension did not occur during the first few hours, after which it proceeded rapidly. The blood stream was always invaded at some time during the course of the infection, within the first hour if the infecting dose was large. No evidence was found that mucin raises the virulence of meningococci. The apparent enhancement of their invasiveness seems to be due to local interference with the defense mechanism of the host." TOOMEY, Cleveland.

TRENDS IN THE PREVALENCE OF DIPHTHERIA. E. L. LUCIA and H. F. WELKE, J. Infect. Dis. **58**:306 (May-June) 1936.

This is an article of importance to all pediatricians and should be read in full. It should be read with caution, since one may mistake statistics for clinical experience. The authors' summary is as follows:

"The statistical evidence presented indicates that the decrease in mortality from diphtheria which occurred in the last decade of the 19th century and in the first decade of the 20th century in San Francisco can not be necessarily attributed to the use of antitoxin.

"Age specific death rates and proportionate mortality rates indicate that children of school age have been most affected by diphtheria as a fatal disease, since 1900.

"In the absence of records it cannot be properly stated that the decrease in morbidity from diphtheria in San Francisco can be ascribed to a program of prevention.

"It is evident that the drop in mortality and morbidity rates in the absence of an extensive official program of immunization, is probably due to a large latent, immune population in San Francisco.

"The present low endemicity of the disease may tend to build up a large reservoir of susceptibles.

"A program of immunization should be undertaken which will make data available concerning the extent of the reservoir of Schick susceptibles and which will build up a reserve of immunes in the susceptible age groups. Such a program would provide for Schick tests both before and after immunization, unless only children under 6 years of age were inoculated." TOOMEY, Cleveland.

THE RACIAL INCIDENCE OF POLIOMYELITIS IN THE UNITED STATES WITH SPECIAL REFERENCE TO THE NEGRO. P. H. HARMON, J. Infect. Dis. **58**:331 (May-June) 1936.

Harmon shows that the incidence of poliomyelitis in white persons in both southern and northern localities is from two to four times that in Negroes. The mortality rates per hundred thousand of population are practically identical. Thus, fatality rates among Negroes with this disease exceed those among the white subjects. There is apparently a lessened incidence of the disease among orientals as compared with white persons in the same area, but this does not seem to be due to racial insusceptibility. It is possible that opportunity for infection by contact is the determining factor here. TOOMEY, Cleveland.

A MILKBORNE SCARLET FEVER EPIDEMIC. Health News **13**:21 (Feb. 10) 1936.

A milk-borne scarlet fever epidemic of 200 cases, with 5 deaths, in a village of 6,000 is reported. Ninety per cent of the patients had taken raw milk or cream purchased from one dealer. The source of infection was found to be a cow with purulent mastitis, which was due to hemolytic streptococci and had followed an injury to one teat and the use of a teat dilator. Two sons of the owner of the farm had previously had sore throat.

AIKMAN, Rochester, N. Y.

PREVENTION AND TREATMENT OF SCARLET FEVER. J. P. KOEHLER, Nebraska M. J. **21**:201 (June) 1936.

In the United States there are approximately 200,000 cases of scarlet fever, with 2,500 deaths, annually. In 80 per cent the disease may be classed as mild.

In controlling the disease quarantine has not been very successful. In Milwaukee, approximately 43 per cent of 3,000 patients who had had scarlet fever were carriers of hemolytic streptococci for from one to five months after their release from quarantine. The tonsillectomized child is as apt to be a carrier as the average child in good health. Children immunized with Dick's scarlet fever toxin show practically the same percentage of carriers as the unimmunized. Release from quarantine by culture is not satisfactory. Immunization by the giving of five doses of scarlet fever toxin is recommended. Those who oppose the use of the toxin are either prejudiced or unfamiliar with it. In the isolation hospital in Milwaukee, since immunization has been done by this method, not a single case of scarlet fever has been reported among the employees during the last eight years. With the approval of the County Medical Society in 1934 a program of immunization was carried out. Three per cent of those immunized had severe systemic reactions; 15 per cent had moderate, 16 per cent mild, 23 per cent local and 43 per cent no reactions. Approximately 64 per cent of the children completed their treatments. Up to April 1, 1936, 8,541 children had had five injections of Dick's scarlet fever toxin. Twenty-four of these had contracted scarlet fever during the past two years. Experience shows that immunized schools are almost free from scarlet fever. Convalescent serum is used for prophylactic treatment and is effective in almost 100 per cent of the cases for a period of fourteen days. Koehler recommends an extensive immunization program for all susceptible children similar to the present programs of immunization against diphtheria and smallpox.

HAMILTON, Omaha.

ENCEPHALITIS FOLLOWING GERMAN MEASLES. E. A. BAUMGARTNER, New York State J. Med. **36**:907 (June 15) 1936.

A 10 year old girl had a convulsion on the second day after she had shown a rash and fever. A positive Pandy reaction was found, and the cell count gave 3 neutrophils, 69 lymphocytes and 28 red blood cells. The Babinski and Oppenheim signs were present, but Kernig's sign was not.

After a severe illness the girl made a good recovery and has shown no complications.

AIKMAN, Rochester, N. Y.

THE THERAPEUTIC VALUE OF VITAMINS A AND D IN MEASLES. H. M. MACKEY, H. M. LINFORD, M. MITMAN and M. H. WILD, Arch. Dis. Childhood **11**:127 (June) 1936.

The effect of giving vitamin D and vitamin D plus A to children with measles was investigated in 697 patients under 13 years of age at the North Eastern Hospital, London. The 697 patients were divided into three groups. The first group served as controls and had only the ordinary diet of the ward; the second group received in addition 3,000 international (U. S. P. XI) units of vitamin D daily as an emulsion of a crystalline preparation of the vitamin (calciferol), and

the third received the same amount of vitamin D with, in addition, vitamin A equivalent to 6 drachms (22.1 cc.) of cod liver oil of a blue unit value of 7.5.

A comparison of the fatality rates and of the incidence of pneumonia, otorrhea, other complications and skin lesions developing in the hospital, as well as of the duration of pyrexia or of the length of the stay in the hospital, affords no evidence of any favorable effect exerted on the course of the disease by either vitamin D or by vitamin D and vitamin A combined. It is pointed out that this negative result may be due to the fact (*a*) that the time of observation (average, under twenty days) may have been too short to demonstrate any effect from vitamin therapy and (*b*) that the treatment may have been started too late, since in 81 per cent of the cases treatment was not started until after the appearance of the rash, say till the fourth day or later in the disease.

KELLY, Milwaukee.

A CONTRIBUTION TO THE EPIDEMIOLOGY OF ACUTE POLIOMYELITIS. HJALMAR WENNERBERG, Brit. J. Child. Dis. **32**:163 (July-Sept.) 1935.

In this paper Wennerberg attempts to show that neither the contact theory nor the water theory appears capable of solving the problem of the dissemination of poliomyelitis but that the theory of dessemination of the disease through insects would clearly explain all hitherto obscure phases of its appearance. A species of mosquito is suspected.

LANGMANN, New York.

APPENDICULAR SYMPTOMS IN THE ACUTE INFECTIOUS DISEASES. G. W. RONALDSON, Brit. J. Child. Dis. **33**:85 (April-June) 1936.

In the literature appendicitis is mentioned as a complication of a number of acute infectious diseases. It is therefore surprising to find that in London at a large hospital for patients with infectious diseases only 2 appendectomies were performed in 20,000 patients and that in another hospital only 1 appendectomy was done in 7,659 patients.

Cases of scarlet fever and measles (prodromal stage) are cited which were examples of "pseudo-appendicitis." Other diseases in which appendicular symptoms may occur are typhoid fever, smallpox (prodromal stage), malaria and mumps. [Rheumatic fever should by rights be included in this list. Abstractor.]

LANGMANN, New York.

CHRONIC MENINGOCOCCAL SEPTICAEMIA. A. M. STEWART-WALLACE, Brit. M. J. **1**:931 (May 9) 1936.

Stewart-Wallace discusses the clinical features, duration and prognosis of meningococcic septicemia on the basis of several case reports. The condition may last for a few weeks or even as long as twelve months, during which time signs of meningeal involvement are usually absent but sometimes are transiently present at the onset. In general, the symptoms are irregular fever, occasional chills, slight elevation of the pulse rate, some leukocytosis, pains in the limbs and various eruptions of the skin.

It is stated that over 75 per cent of the patients recover. Endocarditis or terminal meningitis may be a fatal complication. Blood cultures are difficult to secure and often deceiving.

ROYSTER, University, Va.

A FURTHER ANALYSIS OF THE TYPES OF CORYNEBACTERIUM DIPHTHERIAE. H. S. CARTER, J. Hyg. **36**:147 (June) 1936.

Carter typed 1,614 strains of Corynebacterium diphtheriae; 5.8 per cent were of the gravis type; 65.1 per cent of the intermedius type; 24.1 per cent of the mitis type, and the other 4.96 per cent of type IV, type VI or a mitis-like type. He analyzed the bacteriologic findings and correlated these with the clinical data.

In the gravis group of 89 infections, the fatality rate was 9 per cent; 35 of the patients had mild diphtheria, 17 had cardiac complications, 7 had paralysis,

5 showed albuminuria and 2 were hemorrhagic. Among the 65 cases of types IV and VI there was 1 death. Among 389 cases of infection due to the mitis strains the mortality rate was 1.7 per cent. Among those of infection due to the intermedius, or predominating, type the mortality rate was 7.8 per cent.

Laboratory methods for the identification of the different types are given.

JANNEY, Wauwatosa, Wis.

WHOOPING COUGH: THE DIAGNOSTIC SIGNIFICANCE OF BLOOD COUNTS. NORMAN D. BEGG and MARGARET F. COVENEY, Lancet **2**:1113 (Nov. 16) 1935.

Sixty-five cases of whooping cough were studied in the late paroxysmal or early whooping stage. Leukocytosis was constantly present; each patient was shown to have on an average of 10,064 leukocytes more than was normal for the age. Lymphocytosis was less constant, especially in the children under 6 months of age. The changes in the blood were at a maximum early in the disease and rapidly subsided after the whoop was established.

LANGMANN, New York.

WHOOPING COUGH. NORMAN D. BEGG and MARGARET F. COVENEY, Lancet **1**:82 (Jan. 11) 1936.

A vaccine prepared from recently cultivated smooth strains of Haemophilus pertussis was used. Injections of this vaccine in large doses during the paroxysmal stage neither curtailed the duration of the disease nor ameliorated the symptoms.

LANGMANN, New York.

WHAT IS SCARLET FEVER FOR THE CLINICIAN? F. G. HOBSON, Lancet **1**:417 (Feb. 22) 1936.

The author presents clinical cases to prove that: (1) Erythema is inconstant in infections due to a hemolytic streptococcus; (2) it may be a feature of those due to nonhemolytic streptococcus; (3) though it is more frequent in infections due to hemolytic streptococci, it is a poor guide to the course, prognosis or infectivity of the disease in a given patient, and (4) infections due to hemolytic streptococci with or without erythema are generally highly toxic and highly infectious and have a striking association with sequelae of all kinds. The appearance of erythema is probably a favorable sign.

Notification in cases of scarlet fever as at present practiced serves no useful purpose.

LANGMANN, New York.

DEAFNESS OF CENTRAL ORIGIN FOLLOWING PERTUSSIS. H. GRENET and ANDRÉ BLOCH, Bull. Soc. de pédiat. de Paris **33**:846 (Dec.) 1935.

A girl 4 years old was deaf in both ears. The condition was first noticed when the child was 1½ years old, when she had pertussis, which ran a severe course with high fever for ten days. There had been no otitis media and no convulsions. It is thought that the condition resulted from bilateral labyrinthitis or central encephalitis. No tests were done to lend support to one or the other of these diagnoses.

BENJAMIN, Montreal, Canada.

BEHAVIOR OF THE BLOOD SUGAR CURVE IN DIPHTHERITIC INFECTIONS. E. BENVENUTO, Riv. di clin. pediat. **34**:134 (Feb.) 1936.

Benvenuto plotted the blood sugar curves of sixty-three diphtheritic patients after the ingestion of 1 Gm. of sugar per kilogram of body weight. The blood sugar curve is higher than normal in cases of moderate or severe angina and in cases of croup. It is lower than normal in cases of severe diphtheritic paralysis. In general, the curve is high when there is sympatheticotonia and low when there is vagotonia.

HIGGINS, Boston.

THE VIRUS OF HEINE-MEDIN DISEASE. FAZ TABÍO, Arch. de med. inf. **4**:181 (July-Sept.) 1935.

The filtrable virus of poliomyelitis shows peculiar characteristics. It resists drying, extreme cold and even prolonged freezing, but is rapidly destroyed by sunlight. It will remain active a long time if preserved in a 50 per cent solution of glycerin and protected against sunlight.

The disinfectants which destroyed it best are mercury bichloride, chlorine, hydrogen peroxide, menthol, phenyl salicylate and boric acid. There seems to be a multiple virus, which possibly explains the various forms of infection.

Domestic animals, flies and mosquitoes do not transmit the disease. It is apparently not carried in milk or other fluids, but the virus will live in such fluids. Transmission is by direct human contact. The nasopharynx is the port of entry. The gastro-intestinal tract does not seem to be a fit source through which the infection gains entry.

The best prophylaxis against the disease lies in proper care and treatment of the nasopharyngeal tract.

SCHLUTZ, Chicago.

HEINE-MEDIN DISEASE. ROBERTO VALDÉS DÍAZ, Arch. de med. inf. **4**:191 (July-Sept.) 1935.

Poliomyelitis is an endemic-epidemic disease, affecting especially young children. It attacks persons of any race and social condition, and its epidemics appear in any latitude. The polymorphous streptococcus of Rosenow is not its causative agent. The disease is transmitted by the bearers of germs. It presents many clinical types.

Convalescent serum, in the acute stage; Bordier's treatment, for convalescence, and massage, muscle exercise and orthopedic care are the safest means in the hands of the physician for combating the disease.

SCHLUTZ, Chicago.

POLIOMYELITIS. O. FERREIRA DA SILVA PINTO, Arch. de pediat., Rio de Janeiro **7**:404 (June) 1936.

The subject is reviewed. The author mentions convalescent serum but does not think its use is of as much value in the stage of invasion as hydrotherapy and spinal puncture.

SANFORD, Chicago.

MEASLES IN CHILDREN. E. MÜLLER, Med. Klin. **32**:757 (June 5) 1936.

This is a general article on the subject of measles, suitable for a textbook but containing nothing which has not been written often before.

DOOLEY, New York.

OBSERVATIONS ON A DIPHTHERIA EPIDEMIC OF 1935. R. WERNER, Med. Klin. **32**:868 (June 26) 1936.

Werner comments briefly on an epidemic of diphtheria occurring in Osnabrück, Prussia, in 1935. There were more than a hundred cases. In about half of these the condition was laryngeal; in the remainder, pharyngeal. There was a mortality of 4 per cent among the patients with pharyngeal diphtheria; none of the patients with laryngeal diphtheria died. Complications of one kind or another developed in 49 per cent of all the patients. An effort was made to correlate the leukocytosis and the fever, and it was found that the patients could be divided into two groups: those with a fall in the leukocyte count accompanying the fall in temperature, and those with an elevated leukocyte count persisting for from three to four weeks after the temperature had fallen to normal. This persisting leukocytosis was present in 41 per cent of the patients and was interpreted as an unfavorable prognostic sign.

DOOLEY, New York.

MODIFICATION OF THE SCHICK REACTION BY THE ADDITION OF NORMAL HORSE SERUM. A. STROÉ, M. MARBE, D. HORTOPAN, H. BACALOGLU and A. CORCIOVA, Bull, Acad. de méd. de Roumanie **1**:199, 1936.

These workers demonstrated that a small amount of serum (0.05 cc.) from an old horse with a negative Schick reaction neutralized up to 5 skin test doses of Schick toxin in 80 per cent of sixty-three experiments. A similar amount of serum from a colt, mixed with from 1 to 5 skin test doses of Schick toxin, suppressed the skin reaction in only a small percentage of cases. The interpretation of the work was complicated by some bad local reactions. Another report is to be published.

DIETRICH, Los Angeles.

THE COMBINED EFFECT OF DIPHTHERIA TOXIN AND "GANGRENE" TOXIN IN THE GUINEA-PIG. A. STROÉ and E. BANU, Bull. Acad. de méd. de Roumanie **1**:229, 1936.

Stroé and Banu became interested in the gangreno-ulcerous lesions of the mouth which they observed in patients with severe scarlet fever and malignant diphtheria. From the lesions they consistently obtained anaerobic organisms in association with either the hemolytic streptococcus or the Klebs-Löffler bacillus. Their experiments, which were done on guinea-pigs, involved the injection of various combinations of diphtheria toxin and the toxins of Bacillus perfringens (Clostridium Welchii) and Bacillus histolyticus (Clostridium histolyticum). Using doses of the toxins which regularly killed the controls, the authors were able to save more of their animals when they used combinations of the respective antitoxins than when they used diphtheria antitoxin alone. The work was well controlled, and the results were clearly significant. A more detailed report is to appear.

DIETRICH, Los Angeles.

DIPHTHERIA AND TONSILLECTOMY. K. A. ROMBACH, Nederl. tijdschr. v. geneesk. **79**:5439 (Nov. 23) 1935.

In view of the successful treatment of a diphtheria carrier, a girl 6 years of age, by adenotonsillectomy and the favorable result of this treatment cited in the foreign literature, the author concludes that this method is the most effective way with which to cope with the condition. He esteems adenotonsillectomy to be urgently indicated in the diphtheria carrier and this treatment to be effectual and harmless.

VAN CREVELD, Amsterdam, Netherlands.

SCARLET FEVER. D. HERDERSCHÊE, Nederl. tijdschr. v. geneesk. **80**:510 (Feb. 8) 1936.

Herderschêe presents a clinical lecture on scarlet fever based on his rich experience in Amsterdam, Netherlands. He comments particularly on the epidemiology, diagnosis, complications, prophylaxis and therapy of the disease. The streptococcic etiology is discussed from different points of view.

VAN CREVELD, Amsterdam, Netherlands.

ADVANTAGES AND DISADVANTAGES OF EPIDEMICS OF MILD SCARLET FEVER. R. J. JAMES, Nederl. tijdschr. v. geneesk. **80**:835 (Feb. 29) 1936.

James warns against neglect in cases of mild scarlet fever, in which classic symptoms may be lacking but in which after-effects, especially hemorrhagic nephritis, may occur.

VAN CREVELD, Amsterdam, Netherlands.

Acute Infectious Diseases

RHEUMATIC FEVER IN NORTHERN CALIFORNIA. AMOS CHRISTIE, Am. Heart J. **12**:153 (Aug.) 1936.

Two groups of children were studied to determine the incidence and nature of rheumatic fever in northern California.

The first group consisted of school and preschool children examined at the Cardiac Center of the San Francisco Department of Health. This survey showed

that 50 per cent of all organic lesions are those of congenital heart disease. This is in marked contrast to the 12 to 20 per cent usually quoted in statistics from the East. These facts suggest the lessened frequency of rheumatic infection in this region.

The second group consisted of one hundred and seventeen children who had rheumatic infection. The outstanding feature of this study was the relatively small percentage of children who showed demonstrable cardiac disease.

In other respects the infection bears a remarkable similarity to rheumatic infection elsewhere, as exemplified by the family history, age, seasonal incidence and type of onset and of cardiac involvement.

GIBSON, Chicago.

A NEW APPROACH TO THE THERAPEUSIS OF HEMOLYTIC STREPTOCOCCIC INFECTIONS. RALPH R. MELLON, Proc. Soc. Exper. Biol. & Med. **34**:474 (May) 1936.

It was believed that if virulent organisms could be transformed to their avirulent phase by the host, desirable therapeutic results might be obtained in conditions associated with blood cultures showing growth of the hemolytic streptococcus. Immune horse serums prepared from two anomalous dissociants of a hemolytic streptococcus from a human being with cellulitis were used to reenforce this tendency of the host to change the nature and virulence of the organism. During the past ten years 118 patients with hemolytic streptococcic infection (cellulitis, abscess, erysipelas, erysipeloid, septic sore throat, acute sepsis, mastoid infection with a septic course or some other infection have been treated with antiserums, with a mortality of 12 per cent. Of special interest were the erysipelatous patients, all of whom recovered, and most of whom were in the early age groups. Associated with recovery of the patient is a return of the calcium-phosphorus product of the blood to normal. This product is usually lowered during the active phase of the disease.

HANSEN, Minneapolis.

RABIES: A CONTINUING CHALLENGE. J. N. BAKER, JAMES G. MCALPINE and J. D. DOWLING, South. M. J. **29**:547 (June) 1936.

Rabies appears to be on the increase throughout the world, especially in the southern part of the United States. Texas shows the greatest average annual percental increase in animal heads demonstrated to be infected. Alabama is second. In Alabama, the northern section of the state has been badly infected with rabies, and the disease is rapidly spreading to the southern portion.

The dog is the animal most frequently implicated in the dissemination of rabies, but the cow is also of importance in this connection.

Arm bites, including those on the hands, were most frequent, comprising 56.7 per cent of the total; 29.8 per cent of all bites occurred on the legs and feet, and 6.4 and 2.3 per cent on the head and the trunk, respectively. Multiple bites were noted in 4.7 per cent of the cases.

Of the antirabies treatments administered, 60.9 per cent were given on the strength of a positive laboratory or clinical test carried out on the suspected animal; 34.5 per cent of the antirabies treatments were taken when no diagnosis whatever had been made for the suspected animal. Approximately 91 per cent of the treatments were administered to white persons and the rest to Negroes, making a proportion of 10 to 1. Since 1929, 20,571 Semple treatments have been distributed in Alabama, and two paralytic accidents have been noted.

SCHLUTZ, Chicago.

SUBCUTANEOUS RHEUMATIC NODULES WITH NO CARDITIS. D. J. THOMPSON, Arch. Dis. Childhood **11**:231 (Aug.) 1936.

A case of acute rheumatism in a child is described in which there was an unusually heavy crop of subcutaneous nodules without any evidence of cardiac involvement. The patient died of intercurrent acute appendicitis complicated by metastatic abscesses of the lung. At autopsy the absence of a cardiac lesion was confirmed.

KELLY, Milwaukee.

AGE-INCIDENCE AND SEX-INCIDENCE OF MILK-BORNE EPIDEMICS. R. M. F. PICKEN, Brit. M. J. **1**:1291 (June 27) 1936.

A statistical study of several infectious diseases along the line outlined in the title of the paper is reported.

ROYSTER, University, Va.

UNUSUAL SUBCUTANEOUS RHEUMATIC NODULES. GEORGE DAVISON, Lancet **2**:1057 (Nov. 9) 1935.

A case of rheumatic fever in a boy of 8 years coming on about six months after an attack of scarlet fever is presented. There were typical rheumatic nodules over the occiput, the right acromion, the elbows and knees and the extensor tendons of both hands and feet. The special feature was large nodules over the sacrum.

LANGMANN, New York.

MENINGOCOCCAL SUPPURATIVE ARTHRITIS OF CRYPTOGENIC ORIGIN. W. CAMPBELL and E. C. GREENFIELD, South African M. J. **10**:545 (Aug. 8) 1936.

The bacteriologic features in a case of suppurative meningococcic arthritis (without accompanying meningitis) of cryptogenic origin are described.

GONCE, Madison, Wis.

FEVER AT THE ONSET OF KALA-AZAR. PAUL GIRAUD, Bull. Soc. de pédiat. de Paris **33**:356 (May) 1935.

A boy 16 months old was taken ill with fever, which gradually became remittent and was not present after twenty-one days. Pallor and splenomegaly developed during this period. The blood showed leukopenia. Laboratory tests for kala-azar were negative, but Leishman bodies were found in smears of bone marrow obtained by puncture of the tibia. Intramuscular injections of a preparation of antimony were followed by a rapid cure of the disease.

BENJAMIN, Montreal, Canada.

A MINOR OUTBREAK OF EPIDEMIC MYALGIA IN PARIS. H. VUILLET, Bull. Soc. de pédiat de Paris **33**:657 (Dec.) 1935.

A number of boys in an orphanage were afflicted with a similar illness. The onset was usually accompanied by pain in the epigastrium and thorax and fever. Occasionally a pleural friction was heard. A few had testicular pain. All recovered after a mild course.

BENJAMIN, Montreal, Canada.

CEPHALIC TETANUS WITH PARTIAL FACIAL PARALYSIS. B. WEILL-HALLÉ, AUBERT and RAMBERT, Bull. Soc. de pédiat. de Paris **33**:666 (Dec.) 1935.

A girl 6½ years old was admitted to the hospital because of paralysis of the lower branch of the left facial nerve, paresis of the left hypoglossal nerve and trismus. There was no evidence of a recent wound or scar, and the only possible portal of entry was considered to have been carious teeth. The child made a rapid recovery.

BENJAMIN, Montreal, Canada.

PERSISTENT AND MASSIVE ASCARIASIS ASSOCIATED WITH ANEMIA AND EOSINOPHILIA IN A CHILD EIGHTEEN MONTHS OLD. P. ARMAND-DELILLE, C. LESTOCQUOY and J. BOYER, Bull. Soc. de pédiat. de Paris **33**:703 (Dec.) 1935.

A girl 18 months old presented signs of meningitis. The spinal fluid was normal, and there was marked eosinophilia. A number of round-worms had been expelled. Large doses of santonin were administered repeatedly, and over a hundred of the parasites were recovered from the stools in about two months. The child had a habit of putting earth into her mouth when playing outdoors.

BENJAMIN, Montreal, Canada.

EFFECT OF ANTI-MALARIA DRUGS ON MALARIA PARASITES: PRELIMINARY NOTE. M. SOBKY, J. Egyptian M. A. **19**:234 (May) 1936.

In this preliminary report Sobky considers the following three points: (1) the immediate effects of the three drugs on the number of malarial parasites in a blood film, (2) the minimum amount of each drug necessary to obtain a negative blood film and (3) the percentage of relapses after treatment with any of the three drugs. The drugs studied were quinine, plasmochin and a derivative of acridine. The method of study was to count the number of parasites per hundred leukocytes in a blood film before administration of the drug, again from one to five hours later and again twenty-four hours later. The results showed an increase in the number of parasites on the first examination following the administration of the drug in all instances, but a decrease twenty-four hours later. The smallest dose of quinine necessary to produce a negative result was 1.2 Gm., given in two days to a child of 7 years. She was still free from the parasites four months later. The smallest dose of plasmochin was seven 0.02 Gm. tablets, taken by a boy of 12 years over three days' time, and he remained free from parasites on reexamination seven months later. The smallest dose of the derivative of acridine was five 0.1 Gm. tablets, taken by a boy of 11 years, who was still free from parasites after seven months.

There were relapses in 29.7 per cent of those who were given quinine, in 12.6 per cent of those who were given plasmochin, and in none of ten who were treated with the derivative of acridine. In the discussion of this paper the lack of controls, the small number of cases studied and the method of study are lamented.

MULHERIN, Augusta, Ga.

MENINGITIS AS A COMPLICATION OF WEIL'S DISEASE. W. A. P. SCHÜFFNER and B. WALCH-SORGDRAGER, Nederl. tijdschr. v. geneesk. **80**:3000 (June 27) 1936.

This is a discussion of twenty-five cases of Weil's disease complicated by meningitic symptoms. True meningitis leptospirosa occurred seventeen times, i. e., in 3 per cent of the cases in which icterus was present and in 12 per cent of the cases in which icterus was absent. In cases of true meningitis, therefore, this etiology should be considered. This leptospiral meningitis proved to be benign. In one patient leptospiras of the classic type could be cultivated from the cerebrospinal fluid, and to this type belonged also the leptospiras cultivated from blood and urine in the other cases in question. A further clinical and eventually also a pathologic examination of meningitis leptospirosa is desirable.

VAN CREVELD, Amsterdam, Netherlands.

Chronic Infectious Diseases

THE LAG PHASE IN EARLY CONGENITAL OSSEOUS SYPHILIS: A ROENTGENOGRAPHIC STUDY. N. R. INGRAHAM JR., Am. J. M. Sc. **191**:819 (June) 1936.

Ingraham speaks of the lag period as that period between the time of fetal infection and the development of the earliest demonstrable osteochondritis and periostitic change. He feels that syphilitic osteochondritis, all other things being equal, becomes roentgenographically manifest approximately five weeks after the fetus is infected, and that syphilitic periostitis requires about four months to develop. Therefore the roentgenographic diagnosis of congenital syphilis, to be of greatest value, must rest on the recognition of syphilic osteochondritis, for by the time periostitis has developed the child has been infected for several months and usually exhibits clinical signs of the disease and has positive serologic reactions. A single roentgenogram obtained when the infant is 6 weeks old should disclose the disease in almost every case.

HENSKE, Omaha.

JUVENILE PARETIC NEUROSYPHILIS STUDIES: PHYSICAL COMPLICATIONS, STIGMAS, AND ENDOCRINOPATHIES. WILLIAM C. MENNINGER, Am. J. Syph. & Neurol. **19**:88 (Jan.) 1935.

Of his series of over 600 cases of juvenile dementia paralytica, Menninger found one or more of the syphilitic stigmas in 86 per cent. The specific stigmas and their frequency were: Hutchinson's teeth, 24 per cent; saddle nose, 10 per cent; choroiditis, 5 per cent; interstitial keratitis, 8 per cent; frontal bosses, 11 per cent; high arched palate, 6 per cent; facial asymmetry, 13 per cent; rhagades, 6 per cent, and a mixed group including osteitis, bowed tibia and hydrocele, 21 per cent. Poorly defined disturbances of the endocrine system are frequently associated with juvenile dementia paralytica and usually take the form of genital hypoplasia or body infantilism. Hypoplasia of the genitalia was found in 33 per cent of the males, while amenorrhea was reported in 25 per cent of the adolescent and adult females. Disturbances of the thyroid gland were found only infrequently and seemed to play no rôle in the development of body infantilism in these cases. DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

JUVENILE PARETIC NEUROSYPHILIS STUDIES: TREATMENT. WILLIAM C. MENNINGER, Am. J. Syph. & Neurol. **19**:257 (April) 1935.

Juvenile dementia paralytica is more resistant to treatment than the adult form of the disease. This is due in part to the fact that the infection is of low grade and that there has been a gross disturbance in the protective function of antibody formation in the child. The fact that young tissue is more easily destroyed is also a possible factor. In 20 per cent of Menninger's own series of 43 cases, the condition showed some improvement; in 9 per cent it was arrested, and in 71 per cent it was unaffected. Including 144 cases reported in the literature, the incidence of marked improvement is 14 per cent and of slight improvement 25 per cent. The prognosis would appear to be better when the interval between onset and treatment is short, when the condition develops in or after adolescence, when the basic mental state is good and, in young children, when a period of normal mental and physical development precedes the onset of symptoms.

In Menninger's series of 43 personally observed cases, 7 patients showed some improvement; all these had fever therapy—1, diathermy; 2, typhoid, and 5, malaria. Mercury, arsphenamine and bismuth were ineffective. Three patients received tryparsamide, 1 of whom showed improvement. Of 8 instances of the use of diathermy in the treatment of juvenile dementia paralytica reported in the literature, improvement was reported in 7. Intensive, prolonged treatment with fever, tryparsamide and the heavy metals is recommended as the best therapy.

DAVIDSON, Newark, N. J. [ARCH. NEUROL. & PSYCHIAT.]

INTRAVENOUS USE OF ACETARSONE IN CONGENITAL SYPHILIS. T. B. GIVAN and G. VILLA, Am. J. Syph., Gonor. & Ven. Dis. **20**:275 (May) 1936.

Thirty children with congenital syphilis were given a total of 463 intravenous injections of acetarsone. The average dose was 0.514 Gm. No untoward reactions occurred which required treatment or discontinuance of the medication. There was an improvement in the general condition of every patient, associated with an increase in weight and height. Improvement of active syphilitic lesions occurred in every case in which such lesions were present. The Wassermann and Kahn tests were favorably influenced by the medication in the majority of cases.

REUTER, Milwaukee. [ARCH. DERMAT. & SYPH.]

TUBERCULOSIS OF THE SHAFTS OF THE LONG BONES. C. K. PETTER and J. P. MEDELMAN, Am. Rev. Tuberc. **32**:285 (Sept.) 1935.

Eight cases of tuberculosis of the shafts of the long bones were found in a series of 4,896 patients admitted to a sanatorium. Clinical diagnosis is based on the finding of a definite area of tenderness on pressure with pain on motion of

the part. Eventually, swelling of the soft tissues, and local heat appear, which may go on to the formation of an abscess. General symptoms are absent. On roentgen examination the lesions appear as rounded clearcut areas of decreased density, occasionally loculated. Later the process may be seen to approach the cortex, with the laying down of new bone as the periosteum is reached. Surgical treatment is more satisfactory than conservative management.

EUGENE SMITH, Ogden, Utah.

THE INTRACUTANEOUS TUBERCULIN TEST AND USE OF THE FLUOROSCOPE IN A COUNTY SURVEY. G. W. WEBER, K. M. MURPHY and F. W. HOLCOMB, Am. Rev. Tuberc. **32**:331 (Sept.) 1935.

The authors describe a thorough yet economical plan for a survey of tuberculosis which they believe demonstrates that:

1. The value of the tuberculin test among school children can be greatly increased through the examination of the parents and the epidemiological study of the families in an effort to trace the source of the contact.

2. The use of the fluoroscope reduces to a minimum the cost of carrying on a survey, thus providing a means of detection well within the limitations of the budget of any community.

EUGENE SMITH, Ogden, Utah.

FATALITY FOLLOWING BISMARSEN THERAPY. J. H. SWARTZ, MAURICE M. TOLMAN and HAROLD LEVINE, Arch. Dermat. & Syph. **33**:874 (May) 1936.

The authors report a case of death due to fat embolism following the use of bismarsen in antisyphilitic therapy. The patient had previously received fourteen intramuscular injections of quinine bismuth iodide (0.2 Gm.) and three doses of neoarsphenamine (0.45 Gm.), following which he had itching of the skin and yellow sclera. This therapy was discontinued, and four weeks later tryparsamide in 2 Gm. doses was started. He had twenty-five injections. Then he was given bismarsen, and after the second injection, 0.2 Gm., he stated that he felt "sick" all week but had no definite symptoms. After the third injection he suddenly became weak, pale and nauseated and vomited. He became steadily worse and died within twenty-four hours after the injection.

DULIN, Iowa City.

TUBERCULOSIS SURVEYS AS THEY AFFILIATE WITH THE FAMILY PHYSICIAN. WILLIAM PAUL BROWN, Arch. Pediat. **53**:570 (Aug.) 1936.

Tuberculin tests and roentgenograms of 2,232 college freshmen revealed that 34 per cent of these had already been infected to a degree with the tubercle bacillus.

Brown concludes his report with a list of suggestions for the family physician to follow in the case of all patients who show evidence of having been infected with tuberculosis.

ORR, Buffalo.

THE THREE "P's" OF PHTHISIS. JOHN M. PRESTON, J. South Carolina M. A. **32**: 31 (Feb.) 1936.

Patience, persistence and perseverance are the three P's of tuberculosis from the standpoint of both the patient and the physician. Preston comments on the indications for pneumothorax, phrenicectomy and pneumolysis. The method employed in each is described.

URSTON, Columbia, S. C.

A STUDY OF HOOKWORM DISEASE. G. R. O'DANIEL, J. South Carolina M. A. **32**:151 (June) 1936.

A study of hookworm in Dorchester County, in the southeast portion of South Carolina, was undertaken. The stools of 1,425 persons were examined, and ova

or worms were found in 436, or 30.5 per cent. Varying symptoms and signs were present in persons infested with Necator americanus. Infestation with hookworm persists as a public health problem in the South. Routine examination of the stools of patients of all ages is urged. WESTON JR., Columbia. S. C.

TUBERCULOSIS OF THE SPINE WITH PARAPLEGIA. J. H. KITE, South. M. J. **29**:883, 1936.

Kite compared ninety-six cases of tuberculosis of the spine without paraplegia and twenty cases with paraplegia in order to determine factors which might play a part in the production of the paraplegia. Sex, age, onset of the disease, trauma, extrapulmonary tuberculous lesions and abscess showed practically the same occurrence for the two groups. The region most involved in the paralyzed group was that from the fifth to the ninth thoracic vertebra, while the region most frequently involved in the nonparalyzed group was that from the ninth thoracic to the second lumbar vertebra. The spinal canal is smallest in the midthoracic region. This fact may explain the increased inflammation in the former group.

Paraplegia is seldom due to bony pressure at the point of angulation but is due to pressure on the cord from the products of tuberculous inflammation. The extent of the tuberculous lesion necessary for paraplegia to occur varied from two to twelve vertebrae, with seven as the average. The nonparalyzed group averaged four diseased vertebrae.

If the paraplegia occurs after the onset of the tuberculous lesion in the spine, the prognosis is good. If it comes on late, the prognosis is poor. Patients admitted early after the onset of paralysis have a good prognosis, while those admitted late have a poor one.

Laminectomies in this series have not hastened recovery. Patients with paralysis show a higher mortality than those without paralysis.

SCHLUTZ, Chicago.

ARTIFICIAL PNEUMOTHORAX IN CHILDREN: A RECORD OF FIVE CASES. C. ROLLESTON, Brit. J. Child. Dis. **33**:93 (April-June) 1936.

The ages of the patients when first seen varied from 13 to 16 years. In three of the patients pulmonary tuberculosis was apparently unilateral, and a favorable outcome was obtained with artificial pneumothorax. In the other two, in which the disease process was bilateral, collapse therapy was also performed, but with little or no benefit.

LANGMANN, New York.

MEDIASTINAL GLAND TUBERCULOSIS IN YOUNG CHILDREN. DOROTHY PRICE, Irish J. M. Sc., May 1936, p. 200.

The paratracheal glands were involved in almost all patients with tuberculosis of the lung who died under the age of 2 years. In the follow-up the death rate was higher when both the paratracheal and the hilar glands were involved than when the hilar glands alone were involved. The paratracheal glands are the last line of defense before the organism gets to the thoracic duct and blood stream.

Removal from contact with tuberculosis must be carried out. Primary infection takes place in from three to six weeks following contact.

SNELLING, Toronto, Canada.

SUBACUTE MILIARY TUBERCULOSIS OF THE LUNGS TERMINATED BY TUBERCULOUS MENINGITIS: ANATOMICOCLINICAL STUDY. L. BÉTHOUX, Bull. Soc. de pédiat. de Paris **33**:513 (Oct.) 1935.

A girl 9 years of age had miliary tuberculosis of the lungs. The period from the time she was first examined until she died was three months. The tuberculin test was negative when she was first observed, but before she died tubercle bacilli were found in the spinal fluid. Histologic examination of the lungs showed some

sclerosis, which would indicate that the lesions were old and slowly progressive; many of the granulomas were interstitial in relation to the blood vessels, which was in favor of their dissemination by way of the blood stream; there was little cellular alveolar reaction.

BENJAMIN, Montreal, Canada.

A CASE OF SUBACUTE MENINGITIS DUE TO HEREDITARY SYPHILIS. ROBERT DEBRÉ, JULIEN MARIE and A. BERNARD-PICHON, Bull. Soc. de pédiat. de Paris **33**:425 (June) 1935.

A girl 6 months of age was thought to have tuberculous meningitis. The tuberculin reaction was repeatedly negative, and roentgenograms of the chest were normal. The spinal fluid, which contained over 1,000 white cells, yielded positive reactions to the Wassermann and Kahn tests. After five months of antisyphilitic treatment the infant appeared well except for some psychomotor retardation.

BENJAMIN, Montreal, Canada.

THE MEINICKE REACTION OF THE SPINAL FLUID. ANTONIO SELLEK AZZI and ALEJANDRO DEL FRADE, Arch. de med. inf. **5**:307 (Aug.) 1936.

The Meinicke test was checked against other methods in seventy-two patients. Agreement was found in 64 per cent. In 16 per cent of the cases the Meinicke reaction was positive, while in the other cases it was negative. The latter were cases of children with syphilitic stigmas, or children of antecedents who were carriers of syphilitic stigmas. This would seem to show that the spinal fluid is normal in hereditary syphilis unless the infection is located in the nervous system. There were some cases in which the Meinicke reaction was negative and other tests were positive. The authors believe that their modification of this test is of value.

SANFORD, Chicago.

THE QUESTION OF THE DEVELOPMENT OF MULTIPLE PRIMARY TUBERCULOUS COMPLEXES IN THE LUNG. L. FREY and R. PRIESEL, Arch. f. Kinderh. **108**:171 (June 30) 1936.

Nineteen cases in which children showed roentgenologic evidence of multiple primary tuberculous complexes in the lungs are presented. Each of the histories shows that the source of infection was an advanced pulmonary process in an adult, so that there was the possibility that the child was exposed repeatedly before immunity had time to develop. This explains the presence of multiple tubercles. An exceptionally slow development of immunity cannot be excluded as affording a possible explanation.

CRANE, Chicago.

THE TUBERCULIN TEST IN CHILDREN THOUGHT TO HAVE TUBERCULOUS LESIONS OF THE SKIN. PAULA SEIER, Klin. Wchnschr. **11**:2028 (Dec. 3) 1932.

The tuberculin patch test was given to thirty-four children, from 2 to 15 years of age, among whom there were some with definite tuberculosis of the skin as well as other cutaneous diseases, to see if it could serve as a differential diagnostic test for cutaneous tuberculosis. The conclusion was reached that the test had no value for this purpose.

SILVERMAN, Syracuse, N. Y.

CALCIFICATION DURING HEALING OF A TUBERCULOUS PRIMARY INFECTION. S. ÜSTEL, Röntgenpraxis **8**:382 (June) 1936.

A child first manifested pulmonary tuberculosis by severe hemoptysis at the age of 3 years. The father had previously been proved to be tuberculous, although acid-fast organisms were not demonstrated in his sputum. The child gave a positive reaction to a percutaneous tuberculin test. Roentgenographic examination at the time of the onset of illness yielded findings consistent with early exudative tuberculosis of the upper lobe of the right lung. Despite the unusual and ominous

nature of the introductory manifestations, the child recovered. At the end of three years roentgen examination revealed a bipolar complex, the peripheral component of which consisted of an unusually large rounded shadow of calcium density; in addition there was some residual thickening of the interlobar pleura.

McCUNE, New York.

PATHOGENESIS OF TUBERCULOUS MENINGITIS. CHRISTIANNE RADMANN, Virchows Arch. f. path. Anat. **295**:563, 1935.

Ten brains with tuberculous leptomeningitis were examined with the aim of determining the correctness of Rich's view that in the majority of instances tuberculous meningitis is not the result of hematogenous infection of the meninges but results from the spread of an older cerebral or meningeal focus. The observations in three of the ten brains are held to confirm Rich's contention. In each of these there was an older process with terminal miliary tuberculosis; i. e. there were cerebral tubercles older than the terminal miliary lesions. Four brains showed progressive primary tuberculosis with overwhelming terminal involvement. The author believes that meningitis of this type can be distinguished histologically from that secondary to an older lesion of the brain or meninges. The conditions in the remaining three brains are placed in an intermediate group. In one of these there was a solitary tubercle of the brain, but histologically the meningeal process was held to be like that of the second group. In the other two a primary complex could not be found.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

PATHOLOGY OF PULMONARY EPITUBERCULOSIS. R. RÖSSLE, Virchows Arch. f. path. Anat. **296**:1, 1935.

Rössle would limit the term "epituberculosis" to the form of pulmonary tuberculosis of children in which a relatively large and deep roentgen shadow clears with apparent restoration of the pulmonary tissue to normal; the condition runs a benign course and ends in recovery. An anatomic explanation of the process is wanting because few of the patients followed during life come to autopsy. The clinical explanation based on roentgen findings is that the process is perifocal inflammatory infiltration, which may be tuberculous or nonspecific, in the latter case often being considered tuberculotoxic. Rössle objects to this interpretation because the shadow may be eccentric to the tuberculous focus rather than perifocal, or it may even be in a lobe other than that which has the tuberculous focus. He describes eight cases that give a clue as to what happens and explains some of the less typical outcomes. The roentgen shadow is due to obstructive compression of a large bronchus by a hilar node in the area of the primary complex, with atelectasis of the lung of the occluded bronchus. Smaller bronchi may be occluded by an intrapulmonary node. Clearing of the shadow is due to subsequent aeration of the collapsed lung. In cases in which the disease ran a less typical course bronchial occlusion and atelectasis were caused by tuberculous bronchitis following invasion of the bronchus from a tuberculous focus. In such instances the tuberculous process may be progressive. Another atypical variation may be due to incomplete bronchial obstruction by nonspecific desquamative catarrhal pneumonia.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

THE PROGNOSIS AND TREATMENT OF EXUDATIVE PLEURISY. S. KARLSON, Hygiea **98**:360 (June 15) 1936.

Of 382 patients with pleurisy treated at the Seraphimer Hospital in Stockholm in 1915-1934 and reexamined in 1935, 16 were dead of nontuberculous diseases and 67 (17.2 per cent) could not be located. Of the remaining 299 patients, 183 were examined and 79 sent information about their state of health. The author found that from 26 to 29 per cent had acquired pulmonary tuberculosis (in 11 per cent, active; in from 3 to 6 per cent, healed) and 12 per cent had died from this disease.

WALLGREN, Göteborg, Sweden.

"Tuberculin-Negative" Erythema Nodosum. N. Landorf, Svenska läk.-tidning. **33**:854 (June 12) 1936.

Landorf reports eighteen cases of erythema nodosum in children with no tuberculous lesions detectable and with negative reactions to tuberculin. One of these cases is of special interest: A girl aged 7½ years had typical erythema nodosum but no reaction to 10 mg. of tuberculin, no roentgenologic evidence of changes in the chest and no tubercle bacilli in the gastric lavage water. She was discharged healthy but was readmitted one and a half years later because of a new eruption of erythema nodosum. This time she reacted to 0.01 mg. of tuberculin; she had an epituberculous infiltration in her lungs, and tubercle bacilli were demonstrable in the lavage water from the stomach.

Wallgren, Göteborg, Sweden.

A Case of Clinically Healed Acute Miliary Tuberculosis. G. Sylvan-Karlsson, Svenska läk.-tidning. **33**:855 (June 12) 1936.

A girl aged 4 years fell acutely ill with high fever. Two weeks later a roentgenologic examination revealed miliary tuberculosis of the lungs. Three months after the onset of the disease the fever was only slight and the general condition of the child satisfactory. During the following two years there appeared several other manifestations of tuberculosis: choroidal tuberculomas, pleurisy, spondylitis and tuberculosis of the spleen. A roentgenologic examination of the chest was made once a month. After one year the miliary tubercles of the lungs began to regress, and after another year there remained only lesions belonging to the primary complex, which had begun to calcify. Wallgren, Göteborg, Sweden.

A Case of Congenital Miliary Tuberculosis. Erik Jacobsen, Ugesk. f. læger **98**:418 (May 7) 1936.

A boy was born April 29, 1935, with a weight of 3,300 Gm. at birth. He was healthy during the first eight days; then he had fever and distention of the abdomen. His general condition gradually grew worse, and he died when 17 days old. Postmortem examination showed miliary tuberculosis with primary complexes in the liver, lungs and spleen (acid-fast bacilli demonstrated). The mother had pleurisy at the delivery, and the child was therefore isolated from her immediately after birth. The placenta was normal. Six weeks after delivery the mother died from miliary tuberculosis. Wallgren, Göteborg, Sweden.

Frequency in Man of Tuberculosis Caused by Bovine Bacilli. A. C. Ruys, Nederl. tijdschr. v. geneesk. **80**:364 (Feb. 1) 1936.

Ruys comes to the following conclusions:

In 3 of 115 cases of adults with pulmonary tuberculosis in Amsterdam, Netherlands, and in 10 of 89 cases in the rest of the province of North Holland, tubercle bacilli of the bovine type were cultured. On examination of the fluid obtained by gastric lavage from tuberculous children in various parts of the country, human tubercle bacilli were isolated in 110 cases and bovine tubercle bacilli in 9. Examination of material from persons with other forms of tuberculosis yielded 55 human and 11 bovine strains. The infections with bovine strains are probably due chiefly to the ingestion of raw milk; contamination by contact in the families was not observed. The clinical course in the 33 cases of infections of the bovine type was similar. To isolate tubercle bacilli from the fluid obtained by gastric lavage, guinea-pigs should be inoculated and cultures should be made. Löwenstein's medium gives satisfactory results; for the isolation of bacilli of the bovine type the medium without glycerin is indispensable. To prevent infection with the bovine type, obligatory pasteurization of milk and intensive combating of bovine tuberculosis are recommended. van Creveld, Amsterdam, Netherlands.

COURSE OF TUBERCULOSIS OF THE LUNGS DURING THE FIRST STAGE IN RELATION TO THE AGE OF THE PATIENT. G. BROUWER, Nederl. tijdschr. v. geneesk. **80**:370 (Feb. 1) 1936.

Brouwer presents the results of an inquiry on three groups of persons with the same type of tuberculosis: one of eighteen young children (from birth to 4 years), one of twenty-seven older children (from 10 to 13 years) and one of twelve adults. He endeavors to prove that in all these patients the lesion corresponded with that of Ranke's primary stage; for this purpose he had to show that at the time of infection the patients were anergic. Anergy could be assumed for the young children because of their age. As to the children of school age, whose infection was proved to have been acquired at school, an inquiry about the residents of their homes made the previous existence of anergy probable. As to the adults, sure proof of preexisting anergy could not be given; arguments in favor of it could, however, be supplied. The mild course of the disease in each of the members of the three groups, too, supported the assumption that the infection in the adults could be considered primary. The importance of the relatively favorable prognosis, even for the adult, is pointed out.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Blood, Heart and Blood Vessels and Spleen

ELECTROCARDIOGRAMS DERIVED FROM ELEVEN FETUSES THROUGH THE MEDIUM OF DIRECT LEADS. JAMES D. HEARD, GEORGE G. BURKLEY and C. RUSSELL SCHAEFER, Am. Heart J. **11**:41 (Jan.) 1936.

Satisfactory electrocardiograms were obtained from eleven fetuses by means of the usual technic as applied to the four standard leads. The period of gestation varied from 9½ to 25 weeks. Of the eleven fetuses seven were obtained by hysterotomy, and four were delivered spontaneously.

Left axis deviation was not present in any of the electrocardiograms. In five instances right predominance was present. The sinus node was dominant in all the initial electrocardiograms. Slight sinus arrhythmia was present in four instances. In one subject the auriculoventricular node became dominant a short time before deflections ceased. The conduction time was only slightly shorter than that in the human adult heart.

Minor variations from the pattern of the normal electrocardiogram for the adult appeared in some instances.

GIBSON, Chicago.

A STUDY OF LEAD IV OF THE ELECTROCARDIOGRAM IN CHILDREN WITH ESPECIAL REFERENCE TO THE DIRECTION OF EXCURSION OF THE T-WAVE. HAROLD ROSENBLUM and JOHN J. SAMPSON, Am. Heart J. **11**:49 (Jan.) 1936.

Electrocardiograms were obtained from fifty normal children, varying in age from 1 month to 16 years. These included the usual three leads and a fourth lead with the first electrode (right arm lead) over the second, third and fourth sternocostal junctions on the left and the second electrode (left arm lead) over from the sixth to the eighth thoracic spine on the posterior portion of the chest wall.

These records were compared with sixty-six records obtained from adults in whom there was no evidence of heart disease.

Of the adults 80 per cent showed the characteristic normal inversion to a T_4 wave. Only three adults showed upright T waves in lead IV.

Of the fifty children thirty-two showed upright T_4 waves. In fourteen the T_4 wave was iso-electric, and in two, diphasic. In only two subjects (aged 14½ and 15 years, respectively) was there an inversion of the T_4 wave.

The inverted T_4 wave, which is normal in adult life, is rare in children, while the upright T_4 wave is a normal occurrence in childhood.

GIBSON, Chicago.

THE CHEMICAL NATURE OF THE ANTI-ANEMIC PRINCIPLE. JULIUS SCHULTZ, Am. J. Digest. Dis. & Nutrition **3**:405 (Aug.) 1936.

This is a highly technical review on the studies made to determine the chemical nature of the antianemic principle contained in liver and liver extract.

To summarize, until 1935 most of the investigators believed the antianemic principle to have a nitrogenous base, while Dakin has since shown that a dextrosamine peptide, perhaps derived from a mucin-like substitute, may be the active principle.

MORRISON, Savannah, Ga.

MYELOGENOUS LEUKEMIA IN INFANCY. M. MORRIS and S. HURWITZ, Arch. Pediat. **53**:367 (June) 1936.

Two cases of myelogenous leukemia following pertussis are reported with presentation of the observations at necropsy. In one case the authors mention an involvement of the central nervous system which they feel was produced by the myelogenous leukemia.

ORR, Buffalo.

A FAMILIAL HEMORRHAGIC CONDITION OF AN UNUSUAL TYPE. JAMES B. GILLESPIE, Illinois M. J. **68**:83 (July) 1935.

Gillespie reports two distantly related families tainted with a peculiar type of hemorrhagic condition. Among the 170 members of the two families there were 13 male bleeders, 2 female bleeders and 1 baby boy who was considered a possible bleeder. In 2 members hemorrhage had been the primary cause of death. Laboratory studies were done to rule out the recognized types of hemorrhagic conditions. The studies gave negative results except for a consistent prolongation of the bleeding time in the baby.

BARBOUR, Peoria, Ill.

THE VALUE OF THE PROGNOSTIC VENOM REACTION IN THROMBOCYTOPENIC PURPURA. S. M. PECK, N. ROSENTHAL and L. A. ERF, J. A. M. A. **106**:1783 (May 23) 1936.

The report considers the value of the venom of the moccasin snake in essential thrombocytopenic purpura haemorrhagica. Fifty patients furnished the basis for the data. In a condition of the form of purpura an intradermal injection of this venom causes capillary rupture with diffusion of blood into the tissues. If this occurs after twelve hours, it is considered a delayed reaction. A positive reaction indicates the presence of a purpuric state and a close association with thrombocytopenia. Clinical improvement is manifested by a change from a positive to a negative reaction. Subcutaneous injection of the venom for therapeutic purposes was of value in twenty-two of the thirty-four cases in which it was used. The effects of both the subcutaneous injection and the intradermal test are of importance for the indication and the prognosis of splenectomy.

BONAR, Salt Lake City.

A COMPARISON OF THE ELECTROPHORETIC MOBILITIES AND SEDIMENTATION VELOCITIES OF RED CELLS FROM NORMAL AND PREGNANT HUMAN SUBJECTS. BETTY R. MONAGHAN, CARL WEGNER and H. L. WHITE, J. Clin. Investigation **15**:313 (May) 1936.

Whereas red cells from a pregnant human subject sink in their own plasma from three to twenty-five times more rapidly than do cells from a normal person in their own plasma, no consistent difference is found in the respective velocities of sedimentation in a common medium, 1 per cent gelatin. No measurable difference exists in the electrophoretic mobility of cells from normal persons as compared with cells from pregnant persons in a common medium. It is concluded that the increased velocity of sedimentation of cells in blood from pregnant women is due entirely to changes that occur in the plasma during pregnancy.

AUTHORS' SUMMARY.

RELATIONSHIP OF STRAINS OF GREEN STREPTOCOCCI TO THE CLINICAL CHARACTER OF SUBACUTE BACTERIAL ENDOCARDITIS. HERBERT FOX, J. Infect. Dis. **58**:230 (May-June) 1936.

The fulminating type of subacute bacterial endocarditis may be caused by rough as well as by smooth colonies. Fox suggests that in the cases in which this disease is of the slower type it is probably caused by nonhuman streptococci, more often smooth than not. The significant point in this paper lies in the fact that it indicates that more attention will probably have to be paid to rough colonies, which are now often ignored.

TOOMEY, Cleveland.

THE RELATION OF NUTRITIONAL DEFICIENCIES TO THE DEVELOPMENT OF HEART FAILURE IN ORGANIC HEART DISEASE. W. B. PORTER, J. South Carolina M. A. **32**:145 (June) 1936.

Three cases are cited by Porter, in which the nutritional deficiencies had a direct bearing on the cardiac involvement. A condition which is common in pregnancy is that of hypertension of albuminuria when the diet is so restricted that a sufficient amount of vitamins is not received to prevent avitaminosis of the heart. The author concludes by stating, "The future health of the patient with organic heart disease is definitely influenced by his ability to obtain, ingest and utilize a balanced diet in optimal quantities."

WESTON JR., Columbia, S. C.

DIAGNOSIS OF THE BLOOD DYSCRASIAS IN CHILDREN. RUSSELL L. HADEN, M. Clin. North America **19**:1967 (May) 1936.

The manifestations of the blood dyscrasias are often exaggerated and variable in youth since the hematopoietic system of the growing child is more unstable than that of the adult. Immature cells appear more frequently, and the occurrence of abnormal cells may not have the same significance as in the adult.

Blood dyscrasias in childhood involving primarily the red cells concern almost entirely the anemias. These may be classified clinically as:

1. Those due to increased loss of blood from hemorrhage or from accelerated destruction of red cells (*a*) by toxic factors, such as infection, or (*b*) by rapid removal of these cells from the blood stream in the spleen because the cells are abnormal in shape or size, as in hemolytic jaundice, sickle cell anemia and erythroblastic anemia.

2. Those due to decreased formation of blood from (*a*) quantitative decrease in functioning marrow because of infection or because of replacement of erythrogenic tissue by myeloid tissue as in leukemia; (*b*) quantitative decrease in marrow activity as a result of infection, toxemia, etc.; (*c*) qualitative decrease in marrow function because of lack of the specific elements necessary for the formation of red cells from (*a*) insufficient supply of food, (*b*) chronic hemorrhage and (*c*) deficiency in the erythrocyte-maturing factor.

Leukemia is the most important dyscrasia involving the white blood elements occurring in childhood and is usually of the acute type. The cells involved may be either of the lymphoid or of the myeloid series. The differentiation of the two forms is of little value from a clinical standpoint, although the myeloblastic type probably occurs more frequently than was formerly supposed.

The blood dyscrasias involving primarily the platelets and other coagulating elements at this age are: (1) hemorrhage of the new-born, probably due to deficiency of prothrombin; (2) true hemophilia, in which the fundamental defect seems to be an increased resistance of the platelets to dissolution with consequent deficit in the thromboplastin, and (3) hemorrhage due to a deficiency in blood platelets, or thrombopenia.

The clinical and diagnostic aspects of the various dyscrasias are briefly discussed in this paper.

EUGENE SMITH, Ogden, Utah.

"Spray X-Ray Therapy" in Polycythemia Vera and in Erythroblastic Anemia. F. T. Hunter, New England J. Med. **214**:1123 (June 4) 1936.

Spray roentgen therapy is irradiation of the body as a whole with high voltage roentgen rays. Detailed reports are given in two cases of polycythaemia vera in adults and in one case of erythroblastic anemia (Cooley) in a 7 year old American-born boy of Italian parentage, who has two sisters under treatment for the same condition.

The author's conclusions are: Spray roentgen therapy consisting of 1,000 roentgens given over several weeks' time produced remissions lasting three years in two cases of polycythaemia vera; spray roentgen therapy in small doses in a case of erythroblastic anemia brought about favorable changes in the blood picture and brought about improvement in the patient clinically; this form of treatment appears to be the one of choice in polycythaemia vera and deserves further trial in erythroblastic anemia.

Gengenbach, Denver.

Menorrhagia Occurring at the Onset of Catamenia in a Patient with Thrombopenic Purpura. Arthur Stern, New England J. Med. **214**:1148 (June 4) 1936.

Stern reports menorrhagia occurring with the first menstrual period in a girl 13 years of age, who was known to have had essential thrombopenic purpura for five years.

Treatment consisted in preoperative administration of calcium, iron and vitamins with frequent transfusions and a course of mild radiation applied over the spleen, followed by splenectomy. Splenectomy is the treatment of choice if the condition is chronic, provided the Payne index of operability is satisfactory. Irradiation of the spleen as a palliative or curative measure deserves further investigation.

Gengenbach, Denver.

Ultraviolet Irradiation for Secondary Anemia (Preliminary Report). Richard Kovacs and I. M. Leavy, New York State J. Med. **35**:825 (Aug. 15) 1935.

A group of fifty-nine patients, mostly adults with chronic diseases, showed little improvement in the red cell count or hemoglobin content after exposure to ultraviolet irradiation.

Aikman, Rochester, N. Y.

Sicklemia in the Southwest. W. P. Killingsworth and S. A. Wallace, South. M. J. **29**:941, 1936.

The results of studies of the blood and clinical notes are given on sixty cases of sickle cell anemia without anemia. It is doubtful whether there is a true asymptomatic phase of sickle cell anemia. The relationship of other diseases to the nonanemic phase is not clearly understood. The anemic phase may present the clinical picture of rheumatic fever, tuberculosis, syphilis, acute abdominal conditions and obscure anemias. Because of the high percentage of Negroes who have this blood trait, the study of wet blood preparations is warranted when the diagnosis is considered in a patient of this race. Further study in other racial groups is indicated to prove the importance of sickle cell anemia outside the Negro race.

Schlutz, Chicago.

An Experimental Study of Cardiopericardial Adhesions. Robert M. Hosler and John E. Williams, Proc. Soc. Exper. Biol. & Med. **34**:528 (May) 1936.

Adhesions of the pericardium were produced by operative procedures in dogs, but even after two and a half years no signs of cardiac hypertrophy were found grossly or microscopically. Analyses of 2,600 autopsies disclosed 47 in which extensive pericardial adhesions were observed; in 15 of the cases the heart was normal or smaller than normal, and in 32 there was concomitant heart disease

or vascular disease which could account for the cardiac hypertrophy found. It appears that pericardial adhesions per se, especially adhesions to the diaphragm, do not play a rôle in the production of cardiac hypertrophy.

HANSEN, Minneapolis.

A NOTE ON SO-CALLED "CONGENITAL VARICOSE VEINS." F. P. WEBER, Brit. J. Child. Dis. **33**:102 (April-June) 1936.

"Congenital varicose veins" are really only large veins of developmental origin. The enlargement is analogous to the developmental enlargement of the capillaries in telangiectatic nevus.

LANGMANN, New York.

TREATMENT OF NUTRITIONAL ANAEMIA OF INFANTS. LEONARD G. PARSONS, Brit. M. J. **1**:1009 (May 16) 1936.

Parsons gives a general discussion of the etiology, development, prevention and treatment of nutritional anemia in newly born and young infants.

ROYSTER, University, Va.

HEMOCHROMATOSIS AND HEREDITY. R. D. LAWRENCE, Lancet **2**:1055 (Nov. 9) 1935.

A family of nine is described in which two brothers had proved hemochromatosis. Three other brothers and the mother, but none of the sisters, had some signs of the disease. This suggests that hemochromatosis may be a sex-linked hereditary disease, transmitted by females and affecting mostly males.

LANGMANN, New York.

ENLARGEMENT OF THE HEART. J. PARKINSON, Lancet **1**:1337 (June 13); 1391 (June 20) 1936.

One of the main objects of this article is to stress the roentgenologic method of examining the heart. Parkinson emphasizes the inaccuracy of judgments based on percussion of the heart and says "nothing has been added to medical knowledge or progress through cardiac percussion since the beginning of this century and in the light of x-rays it will shrink into obsolescence."

Slight scoliosis is a common source of apparent enlargement of the heart in childhood.

Hypertrophy and dilatation are discussed at some length.

LANGMANN, New York.

THE CARDIAC OUTLINE. T. S. KEITH, Lancet **1**:1466 (June 27) 1936.

Keith believes that there is a fair degree of agreement between the outline of the heart as seen at autopsy and that obtained in an orthodiagram or a teleroentgenogram.

In carrying out this study he recorded anatomic observations on seventy-eight hearts free from heart disease and seventy that had some form of morbus cordis. The lungs were gently drawn aside to display the whole anterior surface of the heart. On a sheet of glass laid over the heart the outline of the heart was drawn.

Only the data on the normal hearts are taken up in this article.

The anterior surface of the heart mass that corresponded to the shadow seen in the x-ray picture was made up of the superior vena cava, the aorta, the pulmonary artery, the right auricle and the right ventricle; in addition, in nine cases, it included a minute tip of the left auricular appendage, the left ventricle, and, in four cases, a millimeter or two of the inferior vena cava.

The upper part of the left margin of the heart, where it is in relation to the great vessels, is the area where this particular method of research gives the least satisfactory results.

The diagrams show four distinct types of normal hearts: (1) the inclined heart; (2) the horizontal heart; (3) the globular heart, and (4) the square or "conus" heart.

LANGMANN, New York.

GALLOP RHYTHM AND THE PHYSIOLOGICAL THIRD HEART SOUND. CREIGHTON BRAMWELL, Lancet **1**:189 (Jan. 25) 1936.

The physiologic third heart sound is produced by vibration of the auriculoventricular valves, whereas the gallop sound is due to the vibration of the atonic ventricle. Further, the physiologic third heart sound results from an acceleration of the blood flow from auricle to ventricle when the rate of venous return to the heart is increased, whereas the gallop sound is due to the sudden distention of the ventricle when the auricle contracts early in diastole. LANGMANN, New York.

THE ALLERGIC BASIS OF PRIMARY PURPURA IN CHILDREN. C. H. D. BARTLEY, A. DOYNE and C. BELL, Lancet **2**:359 (Aug. 15) 1936.

Twelve cases formed the basis of this study. The authors conclude that though purpura may occur in an allergic person as a result of the ingestion of a suitable allergen, it is nevertheless fallacious to label the primary purpuras as allergic, since the patient often shows none of the attributes of allergy and the course of the disease makes an allergic basis seem improbable.

LANGMANN, New York.

THE TREATMENT OF CIRCULATORY FAILURE. H. W. WUNDERLY, M. J. Australia **1**:840 (June 20) 1936.

In this report Wunderly again calls attention to the works of Wollheim and Warfield regarding the differentiation of circulatory failure. This is divided into plus (or cardiac, or central) and minus (or peripheral) decompensation. The former is characterized by dyspnea, orthopnea, cyanosis of the lips and fingers and an increase in venous pressure and is accompanied by an increase in blood volume. The peripheral failure is characterized by absence of dyspnea and by patchy cyanosis, concentration of the blood with decrease in blood volume, decrease in the blood chlorides and lowering of venous pressure.

Central decompensation is treated along the usual lines with digitalis, strophanthin, diuretics and venesection. Oxygen also may be administered.

In peripheral failure there is emphatic need of increasing the blood volume by means of intravenous fluids. Wunderly favors the use of blood and discusses the indications for the use of acacia, dextrose or saline solution. Also, elevating the foot of the bed and bandaging the limbs are advised. He mentions the use of drugs, such as strychnine, camphor, epinephrine and caffeine, but thinks these to be of lesser importance.

GONCE, Madison, Wis.

THE ANAEMIA PROBLEM IN SOUTHERN RHODESIA. W. K. BLACKIE, South African M. J. **10**:393 (June 13) 1936.

The European resident in southern Rhodesia is exposed to a variety of influences giving rise to anemia, such as malaria, blackwater fever, bilharziasis, the heat of summer, deficiency of iron intake and high altitude. These influences, except malaria and blackwater fever, act slowly, inducing a subclinical condition which manifests itself in increased susceptibility to fatigue and intercurrent infections together with impaired powers of recuperation. The development of these features is so gradual that the victim often regards them as a normal accompaniment of residence in the tropics and makes no attempt to have the condition investigated medically. On the other hand, those in whom anemia is detected respond to suitable treatment, showing that even at this altitude and under the conditions that obtain generally it is possible to restore the blood count to normal values. Apart from taking all customary precautions against malaria, bilharziasis and similar anemia-producing diseases, it is important in this country to take a diet rich in minerals and of high protein value and to insure the ingestion of adequate amounts of sodium chloride (because of excessive perspiration in the summer and consequent reduction in blood chlorides and accompanying reduction in gastric acidity, which in turn results in inefficient absorption of iron).

GONCE, Madison, Wis.

FAMILIAL HEMOLYTIC DISEASE. E. LESNÉ, C. LAUNAY and HUREZ, Bull. Soc. de pédiat. de Paris **33**:445 (June) 1935.

A boy 8 years old had a large spleen, anemia, a subicteric color and slightly increased fragility of the red blood cells. A twin sister and the mother also presented signs which were thought to be due to familial hemolytic disease.

BENJAMIN, Montreal, Canada.

ACUTE JUVENILE LEUKEMIA OF THE LYMPHADENIC AND OSSEOUS TYPE. E. TERRIEN, Bull. Soc. de pediat. de Paris **33**:634 (Dec.) 1935.

A boy 7 years old, thought to have osteomyelitis of the ischium on the left side, was found to have a blood picture resembling that of leukemia. There was fever, and swelling and tenderness were noted about the left ischial body and the pubic ramus. Bilateral exophthalmos and blindness developed and later retention of urine. The latter was believed to be caused by enlargement of the lymph glands or bones which obstructed the flow of urine at the neck of the bladder. A diagnosis of myeloblastic leukemia was made, with the possibility of chloroma. Xanthomatosis was mentioned but not thought likely. Roentgenograms of the bones were not obtained. The boy died two months after the onset of symptoms.

BENJAMIN, Montreal, Canada.

THE MORPHOLOGIC SIGNIFICANCE AND DEVELOPMENT OF MALFORMATIONS OF THE HUMAN HEART. H. BREDT, Virchows Arch. f. path. Anat. **296**:114, 1935.

This is the first of a series of contributions on cardiac maldevelopments based on the material of Rössle's institute since 1931. In it Bredt attempts to evaluate the embryogenesis of certain maldevelopments in the light of their morphology and of current and often controversial theories. In the first section he describes hearts in which two or more anomalies were combined and seeks to determine whether such multiple malformations are a necessary result of some primary failure of development in the sense that a septal defect, for instance, may be secondary or compensatory to stenosis of an ostium, or whether the multiple maldevelopments may be brought about by a factor acting independently on different parts of the developing organ. In the second section he deals with atresias and stenoses of the cardiac cavities and ostia. In the third section he offers a classification of the forms of so-called persistent truncus arteriosus communis, and expresses the belief that when the trunk has a valve of three rather than four segments the anomaly is the result of suppression of the pulmonary artery or aorta and not of simple persistence of an originally single truncus arteriosus. In the fourth section are described some unusual auricular defects, such as an unusual defect of the septum, congenital bands of the interior of the auricles and atresia of the mouth of the coronary sinus. The final section relates to failures of development of the coronary arterial system.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

A CASE OF APLASTIC ANAEMIA. T. OTA, J. Orient. Med. **24**:91 (June) 1936.

An unusual case of aplastic anemia came to the notice of Ota. A boy aged 5 years and 7 months suffered from anemia and consequent loss of appetite. A blood test showed advanced anemia, and an analysis gave: erythrocytes, 606,000; leukocytes, 1,600; neutrophils, 8.4 per cent; lymphocytes, 88.8 per cent, and hemoglobin, 7 Gm. per hundred cubic centimeters. In all, sixteen blood tests were made, and two blood transfusions were given. The child died, however, after two months in the hospital.

He also had buphthalmos, and its presence must be expected in future cases of aplastic anemia.

LESLIE, Evanston, Ill.

A Case of Stenosis of the Splenic Vein (Pylephlebostenosis Splenica). John Rö, Norsk mag. f. lægevidensk. **97**:603 (June) 1936.

A girl aged 10 years had an illness that began with hematemesis following a trauma to the abdomen. The hemoglobin was 28 per cent. She was discharged healthy with hemoglobin 80 per cent. Four years later she suffered a new attack of hematemesis and consecutive secondary anemia. At this time splenomegaly was noted. A roentgenologic examination displayed ectatic blood vessels in the esophagus. After another half year she had pains in the abdomen; splenomegaly persisted, but there were no symptoms of bleeding. Splenectomy was suggested but rejected by the parents. During a new hemorrhage four months later, the patient died. At the postmortem examination there was found a stricture of the splenic vein 5 cm. from the portal vein. The cause of the stricture is unknown.

Wallgren, Göteborg, Sweden.

An Unusual Cardiac Disorder in a Child. J. van Lookeren Campagne and S. van Dok, Maandschr. v. kindergeneesk. **5**:355 (June) 1936.

A girl of 3½ years had an irregularity of the pulse caused by auricular flutter and partial auriculo-ventricular block. There was no other evidence of congenital malformation of the heart. Roentgenoscopy revealed only moderate dilatation of the heart, irregular pulsation of the ventricles and flutter-like movements, especially of the right auricle. On the electrocardiograms, taken at various times, an auricular flutter with a frequency of from 200 to 250 per minute was seen, plus a partial block with variable group forming. The possible pathogenesis and etiology of this disorder are discussed, and the suggestion is made that the underlying condition may be congenital bundle block or vagosympathetic nonbalance.

van Creveld, Amsterdam, Netherlands.

The Disease of Besnier-Boeck (A Rare Form of Reticulo-Endotheliosis). J. J. C. P. A. Roovers, Nederl. tijdschr. v. geneesk. **79**:5541 (Nov. 30) 1935.

The clinical symptoms and histologic changes in a rare case of reticulo-endotheliosis, called by Pautrier the disease of Besnier and Boeck, in a girl 18 years of age are reported. The literature on the subject is discussed. The roentgenographic picture of the lungs in the observed case is compared to that found in cases of miliary tuberculosis and *granulie froide* (Burnard and Savé). The author gave a diet rich in vitamins and injections of calcium and a gold preparation.

van Creveld, Amsterdam, Netherlands.

Diseases of Nose, Throat and Ear

A Retropharyngeal Abscess in an Infant of Six Weeks, Complicated by Pneumonia and Osteomyelitis, with Recovery. F. H. Clark and N. Barysh, Arch. Pediat. **53**:417 (June) 1936.

A case is reported.

Orr, Buffalo.

Acute Ear, Nose and Throat Infections Complicated with Hematuria. Morris A. Weinstein, Arch. Pediat. **53**:515 (Aug.) 1936.

Weinstein reports ten cases of hematuria following infections of the upper respiratory tract.

Orr, Buffalo.

A Promptly Reported Milkborne Outbreak of Septic Sore Throat. Health News **13**:97 (June 22) 1936.

Forty-two cases of septic sore throat occurred in Dryden, N. Y. (population 666) between June 7 and June 11, 1936. The epidemic, which was traced to a milk handler, stopped promptly when pasteurization was instituted.

Aikman, Rochester, N. Y.

TREATMENT OF VINCENT'S ANGINA WITH ACETARSONE. C. H. MAXWELL JR., New York State J. Med. **36**:874 (June 1) 1936.

It is advised to use acetarsone locally as a paste or in glycerin. At the same time it should be given internally. Cleansing mouth washes of sodium perborate or hydrogen peroxide were also used in the cases reported. Large amounts of orange juice are advised.

AIKMAN, Rochester, N. Y.

RECENT ADVANCES IN THE BRONCHOSCOPY OF CHILDREN. CHEVALIER JACKSON, Pennsylvania M. J. **39**:685 (June 13) 1936.

The word "bronchoscopy" has come by common consent to be considered as including not only bronchoscopy but direct laryngoscopy, esophagoscopy and gastroscopy; in other words, it means the examination of the air passages down into the depths of the lung and the food passages to and including the pylorus. All the recent advances in direct laryngoscopy and bronchoscopy warrant the following conclusions:

1. Asphyxia neonatorum is, in a certain percentage of cases, due to an obstruction of the air-way that could have been diagnosed and treated efficaciously by direct laryngoscopic or bronchoscopic methods. It is not infrequently due to bilateral laryngeal paralysis from pressure of a forceps or from excessive traction during labor.

2. A blue baby may have obstruction in the respiratory passages.

3. Congenital partial atelectasis is often overlooked. Every baby should have a roentgen examination of the chest before being discharged from the maternity hospital. If any areas of lung are not expanded, these atelectatic areas should be referred to the watchful care of the pediatrician or the practitioner in consultation with the pediatrician.

4. Residual suppurative disease of the lung lingering after acute pulmonary or bronchial disease should not be allowed to drift into bronchiectasis. Early bronchoscopic aspiration will often turn the tide by restoring the defensive power of the lung.

LOGUE, Williamsport, Pa.

CONGENITAL UNILATERAL OCCLUSION OF THE POSTERIOR NARIS. B. CASTAÑEDA, R. ROCCATAGLIATA and J. GARZONI, Rev. méd. latino-am. **21**:1028 (June) 1936.

A case of congenital occlusion of the right posterior naris is reported, in which operation resulted in a good-sized passage. Such an anomaly is rarely reported.

DWYER, Kansas City, Mo.

IRRITATING COUGH DUE TO INFLAMMATION OF PHARYNGEAL LYMPHOID FOLLICLES —PHARYNGITIS GRANULOSA IRRITANS. R. MODEL, München. med. Wchnschr. **83**:810 (May 15) 1936.

The retropharyngeal lymphoid follicles can cause a persistent cough as illustrated by the case reported by Model. The condition is overlooked unless the pharynx is examined carefully. Cauterization of the follicles with trichloroacetic acid gives relief.

BRAHDY, Mount Vernon, N. Y.

ROENTGENOTHERAPY OF CHRONIC TONSILLITIS. E. D. DUBOVY and E. I. OLSHANOVSKIY, Röntgenpraxis **8**:112 (Feb.) 1936.

This communication emanates from Odessa, U. S. S. R. In a previous publication (Dubovy, E. D., and Olshanovskiy, E. I.: *Röntgenpraxis* **6**:441, 1934) the authors reported that they had observed no effects deleterious to the patient from the treatment of chronic tonsillitis with roentgen rays. In the present paper they review the ultimate results of this type of treatment in 700 patients. The period of follow-up after the last treatment varied between one year and three years and two months. The diagnosis of chronic tonsillitis was based on a history of

repeated sore throat. All patients were examined frequently. Of 254 patients of varying ages, 71.7 per cent were rendered symptom-free, 23.6 per cent were bettered, and only 4.7 per cent were not helped, by treatment. Of 88 children, 66 per cent were "cured," 29.5 per cent were benefited, and 4.5 per cent failed to improve. The technic of treatment is described in detail and a brief bibliography is appended.

McCUNE, New York.

SUPRASCAPULAR BRANCHIAL CYST. H. HAMDI and K. S. ÜGE, Virchows Arch. f. path. Anat. **295**:576, 1935.

A supraclavicular cyst in a boy aged 13 years, which had been evident since the age of 4 years, was surgically removed. From a fistulous opening there had escaped a clear mucoid liquid. Microscopic examination showed the lining of the cyst in places to consist of squamous epithelium; in other places, of ciliated respiratory epithelium, and, in the greater portion of its extent, of gastric mucosa. The cyst is held to have been derived from the first branchial arch at an early embryonic period when the epithelium still had its multiple potencies of forming three different kinds of differentiated epithelium.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

HISTOLOGIC PICTURE OF HYPERTROPHIED TONSILS. A. VON SZÜTS, Virchows Arch. f. path. Anat. **296**:557, 1936.

The tonsils of 200 persons of ages from a few months to 50 years were examined histologically. Hypertrophy was observed most often in the age group of from 5 to 15 years. The process was always associated with evidences of inflammation in the tonsils. The basal cell layer of the epithelium of the crypt underwent hyperplasia. Reticulation and desquamation of the epithelium followed. The reticulum of the tonsil was broken and in part had disappeared. This process is ascribed to the action of ferments liberated by the reticulocytes. The collagenous connective tissue was increased.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

LAW OF SYMMETRY IN HEREDITARY DEAFNESS. B. LANGENBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:223, 1936.

The examination of sixty-one deaf-mutes revealed to Langenbeck that the majority of those in whom the condition is apparently hereditary present typical changes of the organ of hearing. The vestibular apparatus can be stimulated on both sides, and tests with the oto-audion reveal entirely identical remnants of hearing in the two ears. In a smaller group of patients, although the vestibular apparatus responds to stimulus, neither ear has any remnant of hearing. In a third, still smaller group, with retention of vestibular stimulability, the remnants of hearing are largely identical in the two ears, but, particularly in the lower and middle frequencies, there are deviations; these, however, can be explained by former inflammatory disorders. The condition in this third group is really a modification of that observed in the first one. In all three groups, however, the findings can be regarded as the manifestation of a law which the author designates as the law of symmetry of hereditary deafness, because the changes are symmetrical in the two ears. He emphasizes that the tests of hearing should be made with the oto-audion rather than with the tuning fork. He states further that tests on normal persons revealed as a rule completely identical curves for the hearing of the two ears, and he thinks that this makes a law of symmetry in hereditary deafness even more probable. Similar observations in other spheres make it probable that beyond this law of symmetry of hereditary deafness there is a more general law of symmetry of hereditary disorders.

The comparatively rare exceptions to the law of symmetry of hereditary deafness are probably explainable by the intermediate hereditary transmission of two types of hereditary deafness from the parents to the children. The child may have the same intermediate type in both ears, or he may have the paternal type of deafness in one ear and the maternal type in the other. The author thinks that

the law of symmetry is the best criterion of the hereditary character of deafness and that for this reason it is highly important in the enforcement of the law for the prevention of defective offspring.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

TREATMENT OF INFLAMMATORY CONDITIONS OF THE EXTERNAL AUDITORY CANAL WITH BRILLIANT GREEN. I. T. DOROSHENKO, Vrach. delo **18**:479, 1936.

Doroshenko points out the efficacy of brilliant green in the treatment of inflammatory conditions of the external auditory canal of the child.

Brilliant green exerts a marked bactericidal action, particularly on the staphylococcus, and is therefore of great value in the treatment of furunculosis and other inflammatory conditions of the external auditory canal, which occur so frequently in children after bathing, as well as other conditions in which maceration of the epithelium occurs.

Brilliant green is used in a 1 per cent solution in 40 per cent alcohol or in the form of an ointment. A tampon saturated with the ointment or with the solution of brilliant green is introduced into the external auditory canal and left there for a day.

This method of treatment was used on 106 patients from the ages of 1 to 16 years. The maximum duration of the infection was three days in 15 per cent of these patients, while in the remainder the duration was even less. The author strongly advocates this method of treatment.

BODER, Los Angeles.

Diseases of Lungs, Pleura and Mediastinum

INSPIRED FOREIGN BODY IN A NEWLY BORN INFANT. C. L. MORAVEC, Arch. Pediat. **53**:426 (June) 1936.

A 6 week old infant inspired a cinder ¾ by ½ inch (2 by 1.3 cm.) in dimensions. One and a half hours after the administration of epinephrine the patient coughed up the foreign body. No x-ray pictures were taken while the cinder was in the respiratory tract. Afterward x-ray pictures were taken to determine the comparative densities of cinder, coal and stone. The cinder was as opaque as the stone, but almost no shadow was cast by the coal.

ORR, Buffalo.

PNEUMONIA IN CHILDREN. HUGH C. THOMPSON JR., Arch. Pediat. **53**:529 (Aug.) 1936.

Thompson submits a statistical study made on 245 children with pneumonia. Thirty-seven cases were classified as instances of bronchopneumonia and 101 as cases of lobar pneumonia. In 20 per cent of the patients the disease occurred during the first year of life and in 54 per cent during the first three years. Seventy-two per cent of the children with bronchopneumonia were under 3 years of age. Twenty-seven per cent of the entire number of subjects had one or more complications. Empyema existed in 24 per cent of the patients.

The mortality rate was 20 per cent. Thompson considers pneumonia occurring in children over 4 years of age as a relatively nonfatal disease. ORR, Buffalo.

CONGENITAL CYST OF THE LUNG: REPORT OF ONE CASE. C. R. JENSEN, Northwest Med. **35**:222 (June) 1936.

Jensen reports a case of congenital cyst of the lung in a man aged 31. He had been sent to a sanatorium at the age of 13. At that time he felt well and was removed against advice after a two months' stay. At the age of 29, he lost 15 pounds (6.8 Kg.) in one year but regained this by regulating his habits. He is now without symptoms. He shows a small area of tympany with suppression of breath sounds and a few scattered râles over the lower lobe of the left lung. A roentgenogram is appended showing the heart and mediastinum displaced to the right. The cyst does not change in size with respiration. No fluid level has been demonstrated.

SCOTT, Philadelphia.

STAPHYLOCOCCAL PNEUMONIA. A. R. MACGREGOR, Arch. Dis. Childhood **11**:195 (Aug.) 1936.

Ten cases of staphylococcic pneumonia in children are described. In four cases the earlier stages were represented, with serous pleural effusion; in six, the later stage, with empyema. The children's ages ranged from 16 days to 3½ years, but eight were under 1 year of age. In nine cases Staphylococcus aureus was isolated; in one case Staphylococcus was the only infecting micro-organism. In four cases there was a mixed infection. At the earlier stages staphylococcic pneumonia is characterized by massive consolidation of one or more localized areas of one or both lungs, without generalized bronchitis.

Hemorrhage and early onset of suppuration are constant features. Suppuration attacks the bronchi and lymph vessels especially. At the later stage the affected part breaks down into a group of ramifying abscess cavities, one or more of which may burst through the pleura. This gives rise to empyema or pyopneumothorax. The pathologico-anatomic picture indicates that infection reaches the lungs via the bronchi. Aspiration of food is a possible cause in some cases, especially in weak infants. Staphylococcic pneumonia is an entity presenting distinctive pathologic characters.

KELLY, Milwaukee.

LUNG ABSCESS. JAMES MAXWELL, Lancet **2**:413 (Aug. 22) 1936.

Maxwell defines "lung abscess" as "non-tuberculous suppuration with cavitation occurring in the lung tissue." The procedures and conditions which are most commonly associated with pulmonary suppuration appear to be operations in the mouth, pneumonia, a foreign body in a bronchus, bronchial carcinoma and certain abdominal operations. According to some, in the majority of cases the condition results from aspiration of infected material into the bronchial tree; others hold that a septic embolus lodging in a branch of the pulmonary artery is a much more frequent cause. Maxwell holds that both theories may on occasion be correct but that in either case there is commonly some underlying pathologic condition of the lung which affords an opportunity for the commencement of the suppurative process. It is also possible that on occasion the infection reaches the lung by way of the lymphatics.

In the course of his discussion on the diagnosis Maxwell states that needling should be employed only under exceptional circumstances. He holds that in most cases procrastination is the wisest policy. Medical treatment should be tried for at least eight weeks.

Bronchoscopic suction has doubtful value as a routine procedure in the cases in which the condition is acute. The cases most suited for this treatment are the same as those in which there is benefit from postural drainage.

Artificial pneumothorax is limited to those cases in which there is a deep-seated abscess which is draining freely into a bronchus but which proves to be slow to heal.

The indications for operation are chiefly abscesses which are situated toward the periphery, which are chronic and in which satisfactory bronchial drainage cannot be established.

LANGMANN, New York.

SO-CALLED HILUS PNEUMONIA IN CHILDHOOD. A. CARRAU and H. C. BAZZANO, Arch. de méd. d. enf. **39**:354 (June) 1936.

Far from being a closed endeavor, the study of pneumonia in young persons has been greatly accentuated during the past few years, particularly in Russia, Germany, France and North America. Notable contributions have also been made in certain countries of South America, and among these the investigations of Carrau and Bazzano have attracted wide attention.

The present survey is concerned especially with that type of pneumonia in young subjects which involves the region of the hilus and which was first brought to the attention of clinicians by da Rocha in 1931.

It is now universally admitted, however, that while in certain cases central pneumonia is localized at the hilus, the more common evolution of the disease

sooner or later brings it to the periphery. This position of the pneumonic area does not modify the duration, the symptomatology, the prognosis or the duration of the attack. Differential diagnosis becomes extremely important when the localization is aberrant; tracheobronchial adenopathies and tuberculosis must always be kept in mind.

The authors review the clinicoroentgenologic observations in twenty-two cases.

AMESSE, Denver.

A CASE OF PRIMARY CANCER OF THE LUNG IN A GIRL FIVE YEARS OLD. P. LEREBOULLET, P. GARNIER and J. COURTIAL, Bull. Soc. de pédiat. de Paris **33**:502 (Oct.) 1935.

A girl 5 years of age had a bloody effusion in the right pleural cavity. Necropsy confirmed the diagnosis of a malignant neoplasm, which on histologic examination proved to be a small cell epithelioma.

BENJAMIN, Montreal, Canada.

TWO CASES OF PRIMARY CANCER OF THE LUNG IN CHILDREN. J. CATHALA and P. DUCAS, Bull. Soc. de pédiat. de Paris **33**:511 (Oct.) 1935.

A boy 10 years of age and another boy, 4 years of age, had malignant neoplasms involving the lung. In the first boy the condition affected the left lung and extended through the intercostal spaces, causing some bulging of the skin. Biopsy confirmed the diagnosis of a malignant growth of the embryonic epithelial type. In the second boy the left lung was involved, and after a biopsy of an axillary gland a diagnosis of lymphoblastoma was made.

BENJAMIN, Montreal, Canada.

PARTIAL PNEUMOTHORAX, WITH INSIDIOUS ONSET, REMAINING FIXED FOR FIVE YEARS IN A CHILD TWELVE AND A HALF YEARS OLD; PRESENCE OF PLEURAL CALCIFICATION. M. DEGLOS, Bull. Soc. de. pédiat. de Paris **33**:623 (Dec.) 1935.

A boy 12½ years old had a diffuse bronchopneumonia with an area of consolidation at the base of the right lung, which did not clear completely. He continued to have fever and had some expectoration. One specimen of sputum was obtained which yielded tubercle bacilli, but repeated examinations thereafter gave negative results. Inoculation of a guinea-pig with gastric contents obtained during fasting gave negative results. The tuberculin reaction was negative. The boy was sent to the country; some improvement occurred, but three years later a roentgenogram of the chest revealed partial pneumothorax extending upward from the right costodiaphragmatic angle, which was obscured, some pleurodiaphragmatic adhesions and an area of increased density in the base of the right lung. The pneumothorax had occurred insidiously without any symptoms or signs, as the boy was repeatedly observed after the previous roentgenogram was made. His general condition remained good, but the pulmonary condition persisted and was present roentgenographically five years later, when he was examined before entering a college. At this time some areas of calcification were thought to be present on the edge of the pleura bordering on the pneumothorax.

BENJAMIN, Montreal, Canada.

FIBROTHORAX IN CHILDREN. RAÚL PEREIRAS and FERNANDO SALAS, Arch. de med. inf. **5**:221 (Aug.) 1936.

Fibrothorax is rare in children. The authors present one case and the observations at autopsy in another. The etiology is tuberculosis. Every child showing infiltration and pulmonary sclerosis should have serial roentgenograms taken. In such cases of hemothoracic retraction artificial diagnostic pneumothorax should be done to see if the pleura is involved.

SANFORD, Chicago.

SPONTANEOUS PNEUMOTHORAX IN AN INFANT. NILS FAXÉN, Svenska läk.-tidning. **33**:851 (June 12) 1936.

An infant boy, earlier healthy, fell ill with acute pneumonia at 7 months of age. A fortnight afterward he was free from fever, but his general condition grew worse. He had severe dyspnea of an asthmatic type with attacks of cyanosis and collapse. A roentgenologic examination revealed a total pneumothorax on the left side with exudate. The exudate was repeatedly tapped (pus) and eventually disappeared one and a half months later. The pneumothorax then began to decrease, and one month later the infant was discharged in good health.

WALLGREN, Göteborg, Sweden.

Diseases of the Gastro-Intestinal Tract, Liver and Peritoneum

THE FATE OF BACTERIA INJECTED DIRECTLY INTO THE CECAL END OF THE COLON. LOUIS WEINSTEIN, Am. J. Digest. Dis. & Nutrition **3**:397 (Aug.) 1936.

Three dogs were used in this experiment, all having a cecal fistula. There was a minimum of leakage from the intestinal operative site, and all the dogs were in an excellent state of nutrition throughout the course of the experiments. The experiments were carried out under two different conditions. Organisms were introduced into the colon first when the dogs were on a diet consisting of lactose, bread and grains and consequently harbored a large percentage of Lactobacillus acidophilus in their intestines and, second, when the animals were on a strict meat diet and eliminated no demonstrable lactobacilli in their feces.

The organisms studied were Serratia marcescens, Sarcina lutea and Bacillus mycoides. S. marcescens and S. lutea could not be recovered from the feces of animals into the colon of which they were injected through a cecal fistula. This finding was the same whether the intestinal flora of the experimental animals was high or low in its content of L. acidophilus.

B. mycoides could be recovered from the feces of dogs as late as seventy-two hours after injection; when vegetative cells were injected, only vegetative cells were recovered; whereas when spores were introduced, spores were recovered. These findings were apparently independent of the status of the animals' natural intestinal flora.

MORRISON, Savannah, Ga.

DISTAL OR REGIONAL ILEITIS, ULCERATIVE ENTERITIS—NOT AN ENTITY. MOSES PAULSON, Am. J. Digest. Dis. & Nutrition **3**:430 (Aug.) 1936.

This editorial reviews the literature relative to ulcerative enteritis being a clinical entity. The main reason for not considering it a clinical entity is that Bacillus dysenteriae has been recovered in the stools of patients having the condition known as ulcerative enteritis.

MORRISON, Savannah, Ga.

FOOD ALLERGY IN MILD AND SEVERE CYCLIC VOMITING. I. M. HINNANT and L. J. HALPIN, M. Clin. North America **19**:1931 (May) 1936.

The symptoms and the clinical and laboratory findings in cyclic vomiting evidence its toxic origin. The observations of Hinnant and Halpin led them to believe, however, that the attack is precipitated and largely influenced by food allergy. Fifteen of twenty patients carefully studied exhibited clinical manifestations of allergy, such as asthma, eczema, etc. The foods to which these patients were sensitive were those commonly included in the daily diet. In no instance was a single food held accountable, but as a rule one, two or three foods were major irritants. Passive transfer proved to be of great value in testing for food sensitivity, especially in the case of nervous and generally hypersensitive children. All the patients in this group who followed the regimen for allergy have noted excellent or good results.

EUGENE SMITH, Ogden, Utah.

PEPTIC ULCER IN CHILDHOOD, WITH CASE REPORT. FRANK J. WILLIAMS, New York State J. Med. **36**:730 (May 1) 1936.

A 5 year old boy with a negative past history had complained of a recurrent pain in the abdomen for several weeks. The pain occurred at periods of the day or night, especially in the early morning. The temperature was normal; there was no abdominal tenderness, rigidity or distention. There was no vomiting or intestinal disturbance, and no occult blood was found in the stools. Peptic ulcer was suspected, and the diagnosis was verified by the x-ray picture. After the patient had been on a modified Sippy diet for several weeks, complete healing occurred. While peptic ulcer is rare in small children, it should be suspected in cases of recurrent epigastric pain. The x-ray picture is a more reliable diagnostic aid than gastric analysis.

AIKMAN, Rochester, N. Y.

EFFECT OF POSTURE ON FOREIGN BODIES IN THE STOMACH. C. L. MCDONOGH, Brit. M. J. **1**:885 (May 2) 1936.

Attention is called to the fact that in accordance with the position of the child, whether supine, prone or erect, a foreign body such as a coin in the stomach may change its position considerably and thus deceive the examiner to the point that he may miss it altogether. McDonogh feels that any coins having a diameter of 1¼ inch (3 cm.) or less will pass out of the stomach within a few hours if the patient is placed in the prone position, preferably after a meal.

ROYSTER, University, Va.

PYLORIC STENOSIS IN A CHILD TWENTY-TWO MONTHS OLD. PAISSEAU, BOEGNER, BROUET and VAILLE, Bull. Soc. de pédiat. de Paris **33**:361 (May) 1935.

A boy 22 months old was thought to have hypertrophy and stenosis of the pylorus. At operation no pyloric tumor was found, but pylorotomy was performed in the usual manner, allowing the mucous membrane to bulge in the incision. The child continued to vomit, and gastro-enterostomy was performed, after which he was cured. Whether the stenosis was due to cicatricial tissue as a result of a healed ulcer in the prepyloric portion of the stomach or to a congenital intrinsic atresia could not be definitely determined.

BENJAMIN, Montreal, Canada.

A CASE OF DUODENAL STENOSIS IN AN INFANT. G. PAISSEAU, BOEGNER and VAILLE, Bull. Soc. de pédiat. de Paris **33**:367 (May) 1935.

A girl 9 days old was observed. She had attacks of vomiting and was constipated, the former condition having begun soon after birth. She improved, but there were several periods of marked vomiting over a number of months, with intermittent intervals of comparative freedom from symptoms. No operation was performed. She died at 8 months of age, at a time when she was not under observation, and the cause of death was not known. Roentgenographic examination had shown a partial obstruction of the duodenum. Two types of duodenal stenosis are mentioned: primary atresia, to which an infant usually succumbs in the first few days of life, and secondary narrowing, due to some abnormal condition, usually congenital, which may not give rise to symptoms until later in life. The location of the stenosis in relation to the ampulla of Vater on the basis of the presence or absence of bile in the vomitus may be misleading; in the case cited the vomitus was not bile stained during the child's first stay in the hospital but contained bile later.

BENJAMIN, Montreal, Canada.

HYDATID CYST OF THE LIVER: TWO CASES IN CHILDREN, ONE WITH RUPTURE. MARCEL FÈVRE and PIERRE PETIT, Bull. Soc. de pédiat. de Paris **33**:597 (Dec.) 1935.

A boy 14 years old was operated on because of pain in the right upper quadrant of the abdomen which had come on suddenly while he was riding in a bus, when he felt something tear in the region of the liver. The latter was enlarged. A

ruptured hydatid cyst was found. It was successfully removed, and the abdomen was closed with a drain in the incision. The child recovered and was doing well but died two years later of rheumatic heart disease.

A girl 11 years old had an uninfected hydatid cyst of the liver which was easily stripped away and removed at operation. The abdomen was closed without drainage, and the child made an uninterrupted recovery.

BENJAMIN, Montreal, Canada.

A LARGE PRIMARY ABSCESS OF THE LIVER DUE TO STAPHYLOCOCCUS AUREUS. B. WEILL-HALLÉ, AUBERT, RAMBERT and MOUCHETTE, Bull. Soc. de pédiat. de Paris **33**:607 (Dec.) 1935.

A girl 7 years old was taken acutely ill, with symptoms resembling those of pleurisy at the base of the right lung. The liver became markedly enlarged during a period of fifteen days and then remained stationary in size. There was no pain, and the child's general condition was good during a further period of observation of two months. There was no jaundice, and urobilin was found in the urine on one occasion. Emetine hydrochloride and arsenic were given but without effect. There was a progressive increase in the polymorphonuclear leukocytes. Aspiration was performed, and thick pus was withdrawn, from which Staphylococcus aureus was grown. Incision and drainage of the abscess, which was in the right lobe of the liver, was done, and staphylococcus anatoxin was administered. The child recovered. Since no other focus of infection could be found, it is thought that the abscess of the liver was primary.

BENJAMIN, Montreal, Canada.

HYDRO-AERIAL COOING NOISE DUE TO MEDIOGASTRIC SPASM. PIERRE-PAUL LÉVY and JACQUES MÉNÉTRIER, Bull. Soc. de pédiat. de Paris **33**:668 (Dec.) 1935.

A girl 13 years old was obliged to discontinue school because of a cooing noise which originated in her abdomen and could be heard at a distance. This was due to spasm of the stomach, which was dilated and ptosed and gave the appearance of an hour-glass under fluoroscopy. The child had always been emotionally unstable, showing one tic after another, and the gastrospasm responsible for the constant audible noise was apparently another functional manifestation. She was to be treated with sedatives, and attention was to be given to her general health.

BENJAMIN, Montreal, Canada.

TRANSDIAPHRAGMATIC AND SUBDIAPHRAGMATIC STENOSIS OF THE ESOPHAGUS IN A CHILD; EXTRAMUCOUS ESOPHAGOTOMY FOLLOWED BY CURE. M. BOPPE, Bull. Soc. de pédiat. de Paris **33**:675 (Dec.) 1935.

A 13 year old boy, in good health, about six years previously had shown evidence of stenosis of the esophagus, which at times was marked and at other times slight. He was treated by repeated dilation with bougies for two years, after which esophagotomy was performed. The abdomen was opened, and the esophagus was easily reached. A longitudinal incision was made through the wall of the esophagus, above and below the diaphragm, to the mucous membrane, which bulged through the incision. The esophagus was not sclerosed and was not constricted by the diaphragm. The child did well after the operation.

BENJAMIN, Montreal, Canada.

THE USE OF HYPERTONIC SALINE SOLUTION IN INFANTS. G. PAISSEAU, BOEGNER and M. C. VAILLE, Bull. Soc. de pédiat. de Paris **33**:707 (Dec.) 1935.

Infants who were dehydrated as a result of diarrhea and vomiting were given from 1 to 5 cc. of a 20 per cent salt solution intravenously, accompanied by hypodermoclyses of physiologic solution of sodium chloride, lactate-Ringer solution, Ringer's solution and a solution of sodium bicarbonate. They showed a decrease in the alkaline reserve, an increase in the chloride content of the corpuscles, an

increase in the ratio of chloride content of the corpuscles to that of the plasma and an increase in the nonprotein nitrogen. The injections of hypertonic saline solution were frequently followed by reactions, marked by shock and fever, and it is thought that these produced favorable results. Of sixty-one infants treated, twenty-six recovered and thirty-five succumbed; of the latter, all but one were hypothreptic.

BENJAMIN, Montreal, Canada.

HEPATIC INSUFFICIENCY IN CHILDHOOD AND ITS TREATMENT BY THE CURE AT POUGUES-LES-EAUX. CAMILLE BERTON, Progrès méd., May 23, 1936, p. 873.

Berton thinks that many disturbances of childhood, such as indigestion with frequent vomiting, urticaria and eczema, nervous instability, anemia and malnutrition, and respiratory disturbances, such as asthma, may have as a basis a malfunctioning liver. The atmospheric conditions and the waters at Pougues are said to act favorably: First, the carbonates and sulfates in the water stimulate the secretions of the liver and the motility of the stomach; second, the calcium in the water acts favorably on the allergic diathesis; third, the remineralization afforded by the water and the climate are of benefit to the nervous system and to the general state of the patient.

LESLIE, Evanston, Ill.

ENTEROCOLITIS AND APPENDICITIS CAUSED BY ASCARIS AND TRICHOCEPHALUS. FERNANDO SALA PANISELLO, JUAN A. JIMÉNEZ and ANTONIO GUERNICA, Arch. de med. inf. **5**:284 (Aug.) 1936.

Ascaris alone or associated with Trichocephalus can produce pictures similar to that of acute appendicitis. In the authors' study 6.57 per cent of all children with a parasitic infestation were affected by Ascaris, 52.74 per cent by Trichocephalus and 14.12 per cent by both. Examination of the stool should never be omitted in the case of a child presenting colic and symptoms of appendicitis. In the study the authors frequently found an absence of eosinophils in the peripheral blood but found eosinophils in the histopathologic sections. This would speak for the formation of the eosinophils "in situ."

SANFORD, Chicago.

ESOPHAGEAL DIVERTICULA. L. E. C. SCHMIDT, Centralbl. f. allg. Path. u. path. Anat. **65**:33, 1936.

Traction diverticula of the esophagus were present in a 7 year old boy dying of diphtheria. Two small diverticula were located in the anterior wall of the upper third of the esophagus; a larger one was present near the level of the bifurcation of the trachea. Furthermore, there were two islands (each about 2 by 0.5 cm.) of gastric mucosa in the esophagus a short distance below the larynx. Inflammatory changes could not be detected in the walls of the diverticula or in the adjacent tissues. The occurrence of traction diverticula associated with aberrant gastric mucosa in the esophagus points to a congenital origin.

HENEKEN. [ARCH. PATH.]

TREATMENT OF ACUTE ENTEROCOLITIS. W. SCHARPFF, München. med. Wchnschr. **83**:795 (May) 1936.

The stools in cases of enterocolitis should be examined to rule out typhoid fever and dysentery. Vomitus should be examined when there is any suspicion of food or other poisoning. Scharpff treats acute enterocolitis with ½ to 1 ounce (15.5 to 31 cc.) of castor oil followed by one day of rest during which time the patient receives only tea with lemon. Enemas and belladonna are given to relieve tenesmus and pain. The second day the patient is given apple as advised by Heisler and Moro. Banana may be substituted for the apple. On the third day the patient receives toast, zwieback, broth and thin gruel. On subsequent days the diet is more liberal. Bismuth and tannic acid preparations are administered in severe cases.

BRAHDY, Mount Vernon, N. Y.

GEE-HERTER'S DISEASE: A NEW CASE FROM MEDICAL A OF RIKSHOSPITAL. O. K. EVENSEN, Norsk mag. f. lægevidensk. **97**:830, 1936.

A twin 22 years old who was brought to the hospital for carpopedal spasms, was mentally and physically infantile, had a large abdomen, was underweight and had pronounced mechanical and electrical hyperirritability.

During three experimental days 16 per cent of the fat and 10 per cent of the nitrogen administered were excreted by the stool. The blood sugar curve after administration of 50 Gm. of dextrose showed the characteristic low increase.

The blood revealed pronounced hypochromic anemia, which improved fairly well after administration of iron. During the treatment with a calcium preparation and cod liver oil, the blood calcium increased from 6.4 mg. (April 4, 1934) to 9.45 mg. per hundred cubic centimeters (Aug. 29, 1934). The blood phosphorus always kept within normal limits.

TOVERUD, Oslo, Norway.

A CASE OF ACETONEMIC VOMITING IN CHILDHOOD. J. H. LANDWEHR, Nederl. tijdschr. v. geneesk. **79**:5559 (Nov. 30) 1935.

Acetonemic vomiting in a girl of 8 years, with delayed excretion of ketone bodies, was caused, in the author's opinion, by a complicating nephritis. Urobilinuria and a slight decrease of the blood sugar level were present. The author discusses the treatment.

VAN CREVELD, Amsterdam, Netherlands.

TREATMENT OF HYPERTROPHIC PYLORIC STENOSIS. A. W. GRIEP, Nederl. tijdschr. v. geneesk. **80**:824 (Feb. 29) 1936.

Griep describes the operative treatment of twenty-eight infants with hypertrophic pyloric stenosis carried out in the last four years in one of the surgical clinics at Amsterdam, Netherlands. Of the twenty-eight infants, twenty-four were boys. One infant died, death being caused by the narcosis.

VAN CREVELD, Amsterdam, Netherlands.

Nervous Diseases

ANOMALIES OF THE CIRCLE OF WILLIS WITH RESULTING ENCEPHALOMALACIA AND CEREBRAL HEMORRHAGE. O. SAPHIR, Am. J. Path. **11**:775, 1935.

Anomalies of the circle of Willis, with resulting interruption of the circulation between the internal carotid and vertebral arteries, may form the anatomic basis of cerebral vascular disturbances. The recognition of such anomalies is important because they aid in the explanation of cerebral hemorrhage and encephalomalacia on morphologically demonstrable grounds in the absence of occluding lesions of the supplying arteries. In addition to local causes for encephalomalacia and cerebral hemorrhage one must consider the condition of the myocardium and evidence of myocardial failure in the various organs. Three brains are described which revealed anomalies of the circle of Willis involving the posterior communicating arteries and in one case an abnormal origin of the posterior cerebral artery. Two of these brains revealed areas of encephalomalacia and cerebral hemorrhage without the presence of occluding lesions in the supplying arteries. Whereas the posterior communicating arteries are not essential in the maintenance of the circulation of the brain under normal conditions, an unhampered collateral anastomosis between the internal carotid and vertebral arteries is important in instances of diffuse arteriosclerosis of the arteries of the base of the brain combined with beginning myocardial failure. This conception of the origin of these lesions of the brain is based entirely on morphologically demonstrable changes and does not require the assumption of theoretical functional disturbances of the circulation. Perhaps similar anatomic observations may explain anatomic changes elsewhere which now are attributed to functional disturbances.

FROM THE AUTHOR'S SUMMARY. [ARCH. PATH.]

OPHTHALMIC SYMPTOMS IN BRAIN ABSCESS. A. COWAN, Ann. Surg. **101**:56 (Jan.) 1935.

Forty-four cases of abscess of the brain form the basis for this report. Of seventeen cases of abscess of the frontal lobe, exophthalmos, swelling of the lids, chemosis of the conjunctiva and pain occurred in five and orbital involvement without exophthalmos in seven. All these findings occurred on the same side as the abscess. There was more or less restriction of either the lids or the eyeballs or both in fourteen cases, and in three of these the sphincter of the iris was also involved. Frank choking was found in fourteen cases (82.3 per cent) in this group. The average level was about 4 diopters. Of the ten patients with choked disks on whom operation was performed, immediate improvement, which continued to complete recovery, was noted in two, no change until death in four and immediate increase in elevation and severity of the choking after operation in four. In nine cases of abscess of the temporal lobe no involvement of the lids or conjunctivae or evidence of palsy of the extra-ocular muscles occurred. Nystagmus was noted in two cases. Inequality and sluggish reaction of the pupils to light occurred in two instances, with the smaller pupil on the side of the lesion in each. Choked disks were noted in five (55.5 per cent) of the nine cases. All the five patients with choked disks were operated on, with subsidence of the choking in four. Homonymous hemianopic fields were found in three cases. In only one of nine cases of abscess of the parietal lobe was there paralysis of the ocular muscles—involvement of both external rectus muscles. Choked disks were seen in three patients (42.8 per cent). In three of seven cases of abscess of the parietal lobe there were no ocular signs or symptoms, Nystagmoid movement was found in six of seven cases of cerebellar abscess. Choked disks were found in three cases, (42.8 per cent), the same degree of change being present in the two eyes. The average level was 5 diopters. After operation the choking disappeared very slowly. Of four cases of abscess of the occipital lobe, there was continued measurable choking of the disk in three cases, or 75 per cent, and in the fourth case the disk was definitely hyperemic. Two patients had homonymous hemianopia.

One or more ocular signs or symptoms were found in all but six of the forty-four cases of abscess of the brain. However, in only seven cases in the entire series were the defects in the visual field of any localizing value. Choked disks were found in 63.6 per cent of the cases. It is interesting to note that in thirteen the degree of choking remained the same or was even increased after operation.

GRANT, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

POLYNEURITIS: A CLINICAL AND PATHOLOGIC STUDY OF A SPECIAL GROUP OF CASES FREQUENTLY REFERRED TO AS INSTANCES OF NEURONITIS. SHERMAN F. GILPIN, FREDERICK P. MOERSCH and JAMES W. KERNOHAN, Arch. Neurol. & Psychiat. **35**:937 (May) 1936.

The authors reviewed the cases of polyneuritis studied at the Mayo Clinic during the past fifteeen years. The condition is usually preceded by a mild transitory infection. Weakness is the most outstanding symptom. Pain and some sensory disturbance are the most common secondary symptoms. The total protein in the spinal fluid was increased in all cases.

BEVERLY, Chicago.

ENCEPHALOGRAPHY: A REVIEW OF EIGHT HUNDRED ENCEPHALOGRAMS WITH SPECIAL REFERENCE TO SUBDURAL AIR. FREDERICK LEMERE and CLARKE H. BARNACLE, Arch. Neurol. & Psychiat. **35**:990 (May) 1936.

"There is a tendency in the present day literature to diagnose a pathologic condition of the brain on the basis of abnormal encephalograms alone. As the normal variations and technical difficulties of encephalography have never been sufficiently studied, interpretation of organic changes must be made cautiously." The author studied 800 encephalograms.

"Twenty per cent of the encephalograms showed air in the subdural space. The actual presence of air in the subdural space was demonstrated ante mortem (trephine) in one case and post mortem in two cases.

"The entrance of air into the subdural space may be associated with an underlying cortical atrophy or may be due to a technical error. The latter condition is more likely when air in the subtentorial space and collapsed ventricles are also present."

BEVERLY, Chicago.

BRAIN TUMORS IN CHILDREN. J. G. LYERLY, South. M. J. **29**:819 (Aug.) 1936.

Lyerly believes that tumors of the brain in children are probably more common than is generally recognized. The symptoms frequently simulate those of other diseases, and the diagnosis may not be made until it is too late to help the patient and prevent blindness.

Unexplained vomiting, headache and symptoms of enlargement of the head should require a careful study of the eyegrounds and neurologic and roentgen examinations to rule out a tumor of the brain. The finding of a choked disk should always indicate a tumor of the brain until this has been disproved.

While the majority of tumors of the brain in children are glioma, most of them have relatively a low grade of malignancy and can be handled favorably with modern methods of neurosurgical treatment. High voltage roentgen treatment should be given for medulloblastoma, the whole cerebrospinal axis being irradiated.

A tumor in the region of the third ventricle, such as craniopharyngioma, produces hypothalamic symptoms, obstructive internal hydrocephalus, signs of hypopituitarism and impaired vision from chiasmal involvement.

Reports of cases are given illustrating the common pathologic types: medulloblastoma, astrocytoma and spongioblastoma polare, of the glioma group, and craniopharyngioma, of the group of congenital tumors.

SCHLUTZ, Chicago.

MENINGITIS IN THE NEWBORN. W. S. CRAIG, Arch. Dis. Childhood **11**:171 (Aug.) 1936.

Clinical observations from the time of birth are described in connection with twenty-one cases of meningitis in new-born infants. Meningitis is usually part of a generalized septicemia, but it is not always possible to determine whether infection of the meninges has been by the blood stream or as the result of direct extension from other foci. The bacteriologic features of meningitis differ in the new-born from those in older subjects. Meningitis in the neonatal period is frequently the result of infection with Bacillus coli; occasionally it results from infection by organisms of comparative rarity. In the twenty-one cases described B. coli was the causal organism in approximately half that number; in one infant meningitis resulted from septicemia due to an atypical organism of the Salmonella group. Morbid conditions of the skin, subcutaneous tissues or surface mucous membranes were present in fifteen cases. Their presence, especially in premature infants, constitutes a definite risk of meningitis. Meningitis in the neonatal period cannot always be diagnosed. The classic signs of meningitis are often absent. Importance is attached to prematurity and the presence of infection as predisposing factors. Signs of intracranial disturbance occurring after the first week of life should always suggest meningitis; of these ocular signs, sponginess of the fontanel and mental restlessness are the most common. Fever of a few days' duration usually precedes death. Convulsions are rare. The condition must be differentiated from pneumonia in afebrile patients and from intraventricular hemorrhage in the presence of signs arising from increased intracranial pressure. Lumbar puncture is essential for a final diagnosis.

KELLY, Milwaukee.

HEMICHOREA ASSOCIATED WITH A LESION OF THE CORPUS LUYSII. J. P. MARTIN and N. S. ALCOCK, Brain **57**:504, 1934.

The patient, a man aged 65, with sclerotic arteries and a blood pressure of 210 systolic and 130 diastolic, suddenly experienced violent jerking movements first of the left leg and then of the left arm. There were no movements of the face. The movements were irregular, involuntary and of large amplitude and

involved the entire limb. The patient died eight days after the onset, and study of the brain revealed marked atheroma of the basal arteries and a small recent hemorrhage involving the greater part of the right side of the corpus Luysi, part of the anterior and lateral portions being spared. There was also a minute hemorrhage at a slightly higher level in the right side of the thalamus, which Martin and Alcock reject as having any clinical importance.

Von Sántha (1932) claimed that there was a "somatotopical" relationship between lesions of the corpus Luysi and chorea, the anteromedial or oral pole being associated with the face, the middle part with the upper limb and the posterior portion with the lower limb. The observations in the case cited are in accord with von Sántha's suggestion.

In the literature are reported many cases of hemichorea in which the damage has been ascribed to structures other than the corpus Luysi. Wenderowic (1928) found the corpus Luysi relatively intact and attributed the hemichorea to irritation of the internal capsule. Wilson (1929) attributed his case to atrophy of the postcentral gyrus. Lewandowsky and Stadelman (1912) described a large lesion involving the lateral part of the thalamus and the internal capsule; it extended into Forel's field. Zannoni (1908) and Malan and Civalleri (1921) described lesions in the upper part of the thalamus. Fragnito and Scarpini (1926) described softening in the right side of the corpus striatum. Martin and Alcock conclude that there is very little reliable evidence that focal damage to any structure in the brain other than the corpus Luysi or its emergent fibers results in hemichorea. They also conclude that hemiballism following a lesion of the corpus Luysi does not differ from other types of chorea except in intensity.

SALL, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

INTRAVENTRICULAR HAEMORRHAGE IN A CHILD. D. LIVINGSTONE POW, Brit. M. J. **1**:886 (May 2) 1936.

Pow reports a spontaneous intraventricular hemorrhage in a child followed by cerebral symptoms, convulsive seizures and death. The autopsy is also reported.

ROYSTER, University, Va.

VENTRICULOGRAPHY BY OPAQUE INJECTION. E. W. TWINING and G. F. ROWBOTHAM, Lancet **2**:122 (July 20) 1935.

Thorium dioxide was used as the opaque medium, as it is freely miscible with cerebrospinal fluid. Roentgenographic results surpassed those of air injection in the definition and ease with which the whole ventricular system can be shown. From the point of view of the patient's well-being, the method offers the great advantage that the intracranial tension is unaltered. The immediate after-effects of injection of thorium dioxide are milder than those of air.

The authors do not claim that thorium dioxide is the ideal substance.

Alexander, in experimental work on the dog, showed that thorium dioxide caused proliferation of the ependymal cells, which apparently become phagocytic to the thorium dioxide.

LANGMANN, New York.

FIBRILLARY CHOREA OF MORVAN, INFANTILE ACRODYNIA AND PSYCHIC DISTURBANCES. HENRI ROGER and JOSEPH ALLIEZ, Ann. méd.-psychol. (pt. 1) **94**:689 (May) 1936.

In 1898 Morvan described under the name *chorée fibrillaire* a peculiar disease; he had seen several cases. The condition is rare, since Roger and Alliez collected only twelve instances, including four of their own, all reported by French authors. The disease is characterized by fibrillary muscular contractions involving the entire body, paroxysms of profuse sweating, erythema and desquamation of the extremities, severe diffuse pains of contusive type, asthenia, tachycardia, increased blood pressure and psychic disturbances consisting of changes of personality, insomnia, anxiety, irritability and extreme agitation, associated in some instances with acute

delirium and hallucinations. Roger and Alliez compare the symptoms of Morvan's fibrillary chorea with those of acrodynia and point out the nearly complete similarity of the somatic and especially the psychic symptoms in the two conditions. With the exception of fibrillary muscular contractions, usually absent in acrodynia, the essential symptoms are the same—pain in the extremities, desquamation and scalded appearance of the hands, sweating, tachycardia, high blood pressure and psychic disturbances, characterized mainly by changes of personality, depression, irritability, anxiety state and agitation. The mental disturbances in acrodynia are colored by features which are peculiar to the early age of the patients, who are usually children. Thus, the impairment of intelligence and arrest of mental development are likely to be more prominent in association with acrodynia, whereas in Morvan's chorea the intellectual faculties usually remain preserved. The authors suggest that the two conditions belong to the same group of neurotropic virus diseases and possibly are caused by an identical virus. The prominence of psychic-emotional and neurovegetative (sympathetic) disturbances points to a diencephalic localization of the disease process. YAKOVLEV, Waltham, Mass. [ARCH. NEUROL. & PSYCHIAT.]

TICS, OBSESSIONS AND EXCESSIVE STATURE OF ENCEPHALITIC ORIGIN. G. HEUYER, MAIRE and LORSIGNOL, Bull. Soc. de pédiat. de Paris **33**:349 (May) 1935.

A woman 20 years of age showed behavior disorders since the age of 8. She grew very tall but without any obesity. Her expressionless face, sialorrhea, the inclination of the head to the right side and the forward bend of the body gave her the appearance of a person with a parkinsonian syndrome which followed encephalitis, which condition she had at the age of 5 years. The Wassermann reaction of the spinal fluid was positive. BENJAMIN, Montreal, Canada.

BRACHIAL MONOPLEGIA. E. SORREL and SORREL-DEJERINE, Bull. Soc. de pédiat. de Paris **33**:354 (May) 1935.

An 8 year old girl who had paralysis of the right upper extremity, of four days' duration was presented for diagnosis. Tentatively the condition was thought to be due to poliomyelitis. BENJAMIN, Montreal, Canada.

ENCEPHALITIS ASSOCIATED WITH PNEUMONIA: REPORT OF TWO CASES. H. GRENET, P. ISAAC-GEORGES and J. DESMARQUEST, Bull. Soc. de pédiat. de Paris **33**:539 (Dec.) 1935.

A 7 year old girl and a 17 month old boy showed neurologic symptoms and signs, the first at the onset of pneumonia and the second three weeks after recovery from pneumonia. In the former child the neurologic symptoms disappeared soon after signs of pneumonia became manifest, while the latter showed slight weakness of the right foot, with a Babinski sign, and an abnormal gait about two years after the disturbance was first noticed. It is thought that encephalitis caused by pneumonia was present in both cases.

BENJAMIN, Montreal, Canada.

DEHYDRATION AND ACIDOSIS IN A CASE OF ACUTE BASILAR MENINGO-ENCEPHALITIS. L. RIBADEAU-DUMAS and LE MELLETIER, Bull. Soc. de pédiat. de Paris **33**:619 (Dec.) 1935.

A boy 3 months of age recovered from mild erysipelas. Three days later he became very ill and showed convergent strabismus, nystagmus and unequal pupils which reacted to light with hippus. The spinal fluid was normal. The infant showed no improvement, and evidence of marked dehydration accompanied by acidosis developed. Necropsy revealed basilar meningitis. A discussion of the possible nervous control of water and of salt balance in the body is included in the report. BENJAMIN, Montreal, Canada.

TORSION SPASM IN A CHILD. E. LESNÉ and C. LAUNAY, Bull. Soc. de pédiat. de Paris **33**:692 (Dec.) 1935.

A boy 13 years old for eighteen months had suffered from muscular spasm about the spine which produced marked lordosis. He had coarse tremor of the extremities on the left side. He was hardly able to walk or sit up and assumed a position of opisthotonos when in bed. The condition resembled a postencephalitic syndrome of the parkinsonian type, but there was no history of encephalitis. He had pneumonia six months before the onset of muscular hypertonicity. The prognosis was poor. Sedatives and large doses of atropine injected intravenously gave him some relief.

BENJAMIN, Montreal, Canada.

ACUTE PRIMARY ENCEPHALITIS (NOT EPIDEMIC) IN CHILDREN. A. FIANO, Riv. di clin. pediat. **34**:97 (Feb.) 1936.

Fiano has assembled twenty cases of acute primary encephalitis from the records of the Institute of Clinical Pediatrics of the University of Florence from 1895 to the present. Cases of polioencephalitis and epidemic encephalitis are not included.

In the acute phases, fever, vomiting and, above all, generalized convulsions were the outstanding symptoms. The cases reported are only those in which the diagnosis could be clinched by the occurrence of sequelae; the outstanding involvement was of the motor system and the intelligence.

HIGGINS, Boston.

ENCEPHALOGRAPHY AND INFANTILE ENCEPHALOPATHIES. J. M. SALA GINABREDA, An. de pediat. **19**:433 (July-Aug.) 1935.

Brisand included in this group both the cerebral lesions originating before birth and the disturbances of cerebral development which clinically show grave intellectual defects, such as idiocy or imbecility, and frequently are accompanied by organic syndromes, such as infantile cerebral hemiplegia, diplegias and Little's disease. Study by encephalogram and ventriculogram determines the limits reached by a disturbance of cerebral development and enables one to give a prognosis more or less favorable, as well as to establish a relationship between the roentgenographic alteration and the intellectual capacity of children with this condition.

Practice teaches that grave mental alterations are not always accompanied by important macroscopic alterations, since the disturbance of development can be entirely histologic and microscopic and not visible by roentgenography. It is more frequent, however, in the encephalopathies of infants to find important roentgenographic alterations, the most commonly observed being the dilatation of one or both ventricles, as well as irregularities in their form. It is not possible to establish a direct relation between the roentgenologic lesions and the intellectual capacity of the child, since this depends ultimately on a series of causes and mechanisms which the roentgenogram cannot reveal.

A difficult point to appreciate in the interpretation of the plates is the existence of slight ventricular dilatations. With reference to the lateral ventricles, the frontal views must be minutely examined; as a basis for the determination of slight, but pathologic, dilatations the following points are taken: 1. The maximum transverse diameter of the ventricular image coincides normally with the superior external angle; that is, a horizontal line from the superior external angle to the internal border of the ventricle gives approximately the maximum diameter of the ventricular image. This diameter is normally always less than one third of the maximum transverse hemidiameter of the skull. 2. The superior external angle of the ventricular image tends to disappear and take a round aspect, losing the characteristic angulation. 3. The external border tends to be concave toward the cavity, instead of convex as normally. 4. The lateral portions of the ventricles are dilated. Histories of cases, with encephalographic plates, are appended.

GIFFEN, Toledo, Ohio.

MENINGITIS. OCTAVIO FERREIRA DA SILVA PINTO, Arch. de pediat., Rio de Janeiro **7**:357 (May) 1936.

All types of meningitis, including meningeal tumors, are discussed in twelve pages—an interesting résumé.

SANFORD, Chicago.

THE CEREBROSPINAL FLUID. MILTON SALGADO BASTOS, Arch. de pediat., Rio de Janeiro **7**:480 (Aug.) 1936.

This is a concise résumé of the ordinary laboratory findings.

SANFORD, Chicago.

THE REPEATED MINOR CONVULSIONS OF CHILDHOOD (PYKNOLEPSY). CURT ROSENTHAL, Ergebn. d. inn. Med. u. Kinderh. **48**:76, 1935.

The first cases of this condition were described by Berkhan in 1892, and in 1896 Furstner termed it "hysterical convulsions in children." The attacks consist of periods of incomplete loss of consciousness, lasting from ten to twenty seconds, thus strongly simulating epilepsy (petit mal). Speech is usually interrupted, but mechanical actions are frequently continued. The loss of consciousness is usually incomplete, and many patients can tell what happened to them during an attack. After the attack is over the patient usually continues the act which it interrupted. Ocular symptoms, such as staring and twisting the eyes and especially rolling the eyes upward, are seen in about three fourths of the patients. Light motor phenomena, such as fluttering of the eyelids, nystagmus, a slight twitching of the face and of the upper or lower extremities, letting the arms fall or holding the body stiff and stepping forward or backward, have all been observed. Coarser motor changes, such as clenching the fist, rhythmic tremors of the extremities, twisting of the head and changes in the facial expression, are also seen. Frank tonic or clonic movements do not belong to the domain of pyknolepsy. Such vasomotor phenomena as slight reddening or blanching of the face have been noted. Loss of control of the sphincter of the bladder and falling over may occur; the latter, however, is always slow, thus accounting for the rarity of sustained injuries.

The other symptoms to be mentioned may as well occur in genuine petit mal as in pyknolepsy. A few of these are respiratory changes, emotional facial expressions, particularly those of fear, and amnesia. An aura or postparoxysmal symptoms are rare. The extraordinary frequency of the attacks should be emphasized. An attack rarely lasts over one minute. The explosive onset of the attacks must be further mentioned; i. e., from the very beginning the number is large, and the attacks are monotonous and periodic. Quite characteristic of this illness is the unimpaired mental and intellectual development. Any patient suffering with these multiple and frequent seizures who shows signs of mental degeneration must be considered to have epilepsy rather than pyknolepsy. Other characteristic symptoms of pyknolepsy are its lack of response to bromides and phenobarbital and also the fact that the attacks are uninfluenced by extrinsic physical or emotional factors.

The disease usually begins in childhood and terminates around puberty, although occasionally it may extend into the third or the fourth decade of life. Physically the patients show nothing characteristic; likewise, intellectually they do not deviate from the normal.

The average age of cured patients is from 10 to 11 years; the average duration of the pyknoleptic phase is three and a half to four and a half years. The incidence of this disease is slightly greater in girls than in boys. In cases of pure pyknolepsy the outlook as far as complete recovery is concerned is good; in those of the "mixed form" (conditions that had been diagnosed as pyknolepsy and later found to be true epilepsy), however, the prognosis is much less favorable. In a study of 102 cases (66 from the literature and 36 of the author's), the author found evidence of a hereditary epileptic taint in 18 per cent, a frequency somewhat similar

to that of true epilepsy. Therapeutically, there is nothing entirely satisfactory for this condition, certainly nothing that compares with bromides and phenobarbital in the treatment of epilepsy. Nutritious diet, prevention of overfatigue, enough sleep, regulated digestion, sufficient fresh air and occasionally a change in the milieu and sometimes psychotherapy are of some help.

CAPPER, Philadelphia.

BILATERAL TUMOR OF THE AUDITORY NERVE AS A COMPONENT OF GENERALIZED NEUROFIBROMATOSIS. L. KRAUS, Fortschr. a. d. Geb. d. Röntgenstrahlen **53**:793 (May) 1936.

A 9 year old girl had exhibited the multiple tumors characteristic of Recklinghausen's disease since 1 year of age. At 18 months she commenced to display evidence of progressive impairment of hearing, and this was followed some years later by mental deterioration. By 9 years she was almost completely deaf. Roentgen examination revealed changes in the contour of the porus acusticus internus on both sides; biopsy confirmed the neurofibromatous nature of the skin tumors and, by inference, established securely the cause of deafness.

Neurofibromas of the eighth nerve are usually bilateral. Similar cases have been reported previously; the peculiar importance of this one lies in the fact that the diagnosis was confirmed by the x-ray picture.

McCUNE, New York.

MALARIA TREATMENT OF NONSYPHILITIC NERVOUS DISORDERS IN CHILDHOOD. V. L. DOBSZAY and I. FISCHER, Ztschr. f. Kinderh. **57**:124 (March 21) 1935.

One patient with severe epilepsy and mental deterioration showed remarkable improvement after malaria treatment. Two patients with early postencephalitic disturbances recovered completely. One with Little's disease and two with idiocy seemed slightly improved. Five patients (two with late status postencephaliticus, two with idiocy and one with epilepsy) showed no improvement. One patient with Little's disease died. In one case of late status postencephaliticus serious complications necessitated termination of the treatment.

Since the treatment is not without some danger, it should be used only in cases in which the slightest prospects of improvement outweigh the risk involved.

SIEMSEN, Chicago.

A CASE OF EPILEPSY COMBINED WITH CALCIFIED INTRACRANIAL HEMANGIOMA. E. KYLIN and T. KJELLIN, Acta med. Scandinav. **88**:107 (Feb. 28) 1936.

A case of Sturge-Weber's syndrome is reported. The 17 year old patient, who had had idiopathic epilepsy since the age of 5 years, had no vascular nevus of the face, but roentgen examination revealed a tortuous, wormlike calcium deposit in the occipital region of the cortex. The vasomotor system was involved, and deterioration of the memory and changes in the personality were developing.

In forty of the eighty-seven cases reported in the literature the patient had symptoms in the first year of life.

KAJDI, Baltimore.

CEREBRAL TRAUMA IN CHILDREN. J. J. G. PRICK, Maandschr. v. kindergeneesk. **5**:324 (May) 1936.

Prick reports ninety-nine cases of acute trauma of the cerebrum in children observed between March 1932 and October 1935 and the lines along which at the moment the therapy of such conditions is conducted in the Neurological Clinic of the Wilhelmina Gasthuis at Amsterdam. In order to illustrate these lines, three interesting clinical observations are mentioned; they show the good results of the therapeutic measures which are taken on correct indication. In the greater part of the cases conservative treatment is preferable, even in those cases in which temporary severe symptoms of irritation and defect make the clinical picture serious.

VAN CREVELD, Amsterdam, Netherlands.

APHASIA IN A GIRL FOUR YEARS OLD. C. T. VAN VALKENBURG, Nederl. tijdschr. v. geneesk. **80**:827 (Feb. 29) 1936.

Valkenburg reports in detail the case of a girl 4 years old in whom aphasia followed a traumatic lesion of the left temporal lobe. He discusses exhaustively the restitution of unimpaired speech after complete mutism—with good understanding—without a falling back to an earlier level. The difference between aphasia in little children and in adults is emphasized in connection with the normal development of speech. VAN CREVELD, Amsterdam, Netherlands.

Psychology and Psychiatry

MONGOLISM IN ONE OF DIZYGOTIC TWINS: REPORT OF A CASE. R. L. JENKINS, Illinois M. J. **69**:455 (May) 1936.

Jenkins reports a case of mongolism in one of dizygotic twins. The fact that the other twin was normal, he believes, makes untenable any etiologic hypothesis which depends on a humoral mechanism of the maternal blood.

BARBOUR, Peoria, Ill.

HYSTERIA IN CHILDREN. AGUSTIN M. ABRILL, Arch. de med. inf. **5**:247 (Aug.) 1936.

A case is presented to show that such an entity as hysteria exists in children. Abril believes that Babinski has underestimated emotion as a cause of hysteria. It may well be a factor, although not often. Sexual problems often provoke hysterical accidents. Psychoanalysis is the treatment of choice.

SANFORD, Chicago.

MONGOLIAN IDIOCY IN TWINS. HANS HELLSTEN, Monatschr. f. Kinderh. **66**:400 (Aug.) 1936.

Hellsten reports two cases of mongolian idiocy occurring in two pairs of twins in which the other twin sibling was normal. There is also a brief discussion of the cases already reported in the literature. GERSTLEY, Chicago.

UNUSUAL ARTISTIC ABILITY IN A CHILD. FRANZISKA BAUMGARTEN, Ztschr. f. Kinderpsychiat. **2**:182 (Feb.) 1936.

The case of a child gifted with unusual drawing ability is reported. At as early an age as 3 years the boy was able to reproduce pictures and caricatures of animals and persons with minute detail. Drawings made at different periods in the child's life are reproduced, and Baumgarten discusses some psychologic phases of various details depicted. Though of normal, uneventful physical development, the boy died early in youth. JAHR, Omaha.

Diseases of the Genito-Urinary Tract

TUBERCULOSIS OF THE KIDNEY IN CHILDHOOD AND ADOLESCENCE. HERMAN L. KRETSCHMER, Illinois M. J. **70**:119 (Aug.) 1936.

This paper consists of a review of a series of forty-three cases of renal tuberculosis in children and adolescent persons. The article is too comprehensive to abstract completely. Some of the more important points presented by Dr. Kretschmer are as follows: The question of sex is not an important aid in the diagnosis of renal tuberculosis. A healed tuberculous lesion or perhaps the presence of an active one should arouse suspicion, in the case of a patient with urinary symptoms or in the case of a patient who persistently has pus in the urine but no urinary symptoms, that the patient may be suffering from a tuberculous lesion in the urinary tract, and hence one should direct the examination with

the possibility of renal tuberculosis in view. Evidence of tuberculosis elsewhere than in the renal system was obtained in 72 per cent of this series of cases.

One of the most important problems in every case of renal tuberculosis is to determine the exact status of the opposite kidney with regard to freedom from tuberculosis, as well as to its functional capacity.

The end-result in the case of a patient for whom the diagnosis is made early who has only a moderate amount of involvement of the bladder or perhaps no involvement of the bladder is much better than that in the case of a patient in whom the diagnosis is made late and the tuberculosis of the bladder is extensive.

Urinary symptoms were present in the largest number of the cases; they tended to increase in severity and failed to respond to medical treatment. Of the various urinary symptoms encountered, frequency of urination heads the list, having been present in 86 per cent of the cases. The frequency in most cases is due to the presence of tuberculous ulceration of the bladder and generally means that the renal tuberculosis has been present for a long time. In some cases frequency may be due to the fact that the patient has a contracted bladder; hence a limited capacity requires frequent emptying. In some of the early cases without involvement of the bladder the frequency may be due to tuberculous pyelitis. In other cases the frequency may be due to polyuria. Persistent nocturia should always arouse suspicion, especially if the urine contains pus.

The most frequently encountered positive test resulted from albumin in the urine. Blood—either gross, in chemical combination or in the form of red blood cells—was found in thirty-three cases. Pus occurred in forty-one of the forty-three cases. Examination for tubercle bacilli should be carried out as a routine in every case of persistent pyuria. In cases of renal tuberculosis roentgen ray examination of the urinary tract in the largest number of cases gives negative results.

The cystoscopic findings in cases of tuberculosis of the bladder secondary to renal tuberculosis are so characteristic that the diagnosis of tuberculosis is self-evident. Catheterization of the ureters is carried out for two purposes: (1) to demonstrate the origin of the tubercle bacilli found in the mixed specimen, whether from one or from both kidneys, and (2) to determine the functional capacity of the normal kidney.

The treatment of unilateral renal tuberculosis is surgical, after the opposite kidney has been carefully studied as regards its freedom from tuberculosis and its functional capacity. Bilateral chronic renal tuberculosis is not a surgical condition.

BARBOUR, Peoria, Ill.

GONORRHEAL VAGINITIS. R. M. LEWIS and E. L. ADLER, J. A. M. A. **106**:2054 (June 13) 1936.

Estrogenic substance in ethylene glycol given hypodermically was relatively effective in treating gonorrheal vaginitis when given in large doses, 2,400 international units daily. Smaller doses proved disappointing. Vaginal suppositories of the substance in 600 and later 1,000 unit doses were very effective. Cessation or diminution of the discharge appears in from fourteen to eighteen days. The authors believe that a change in the reaction of the vaginal secretions from neutral or alkaline to acid is the major factor in eliminating the infection. The degree of acidity of the secretion offers a guide as to the adequate dose. Of thirty-three consecutive patients in whom this manner of treatment was followed thirty yielded negative smears in three weeks. Two required twelve weeks. Five patients were listed as showing recurrences. No ill effects were noted. This treatment is believed to be the most effective known for the treatment of gonorrheal vaginitis in children.

BONAR, Salt Lake City.

THE AMMONIACAL DIAPER. D. O. RHAME JR., J. South Carolina M. A. **32**:153 (June) 1936.

Excoriation from ammoniacal diapers is a common manifestation. It exists frequently in constipated children and is often associated with a diet consisting of milk, cereal, bread and much fat. Rhame discusses the metabolism of ammonia

in the urine. It is the free, not the combined, ammonia which causes the irritation of the skin. Occasionally there exists bacterial decomposition of the urea, which produces the so-called diaper rash. However, Zahorsky has shown that improper cleansing of the diapers may be a cause. Either the diapers are washed with a strong alkaline soap, or they are improperly washed or cleansed. The ammonia which is derived from the ammonium definitely is released by the alkali in the diaper from soap, lye, stool or lime.

WESTON JR., Columbia, S. C.

HYPERNEPHROID TUMORS OF THE KIDNEY. E. R. WHITMORE, South. M. J. **29**:1051, 1936.

Whitmore reports three cases of hypernephroid growth of a tumor of the kidney or of the adrenal gland with and without metastasis. In one of these, the clinical picture was that of an acute ruptured hemorrhagic cyst of the kidney. Another patient showed a tumor present which had metastasized before operation. In the third, pulmonary symptoms dominated the clinical picture. At necropsy there were a tumor in the left kidney, nodules in both kidneys and metastases in the lymph nodes and lungs.

Between 90 and 95 per cent of tumors of this sort are radiosensitive. Surgical removal and radiation therapy offer the best results for relief or cure.

SCHLUTZ, Chicago.

URINARY TRACT COMPLICATIONS IN THE PROLONGED IMMOBILIZATION OF CHILDREN. L. A. KEY, Brit. M. J. **1**:1150 (June 6) 1936.

Key calls attention to the high incidence of infection of the urinary tract in children who are under prolonged treatment requiring immobilization. He points out the noticeable lack of manifestation of leading symptoms and signs and makes a plea for early and complete investigation.

ROYSTER, University, Va.

RENAL FUNCTION TESTS IN CHILDHOOD. J. H. HUTCHISON, Glasgow M. J. **7**:257 (June) 1936.

Hutchison describes and compares the urea concentration test and the urea clearance test. He states that the urea concentration test is misleading, especially if the subject drinks a large quantity of water and passes dilute urine. The urea clearance test, on the other hand, depends on complete emptying of the bladder at stated intervals. This is not always possible in children, as they sometimes refuse to urinate, and unless one uses the catheter one cannot be sure that evacuation of the bladder has been complete. If this objection can be overcome, the latter test is the more accurate measure of renal function.

SNELLING, Toronto, Canada.

UROLOGY IN CHILDREN. O. L. ADDISON, Proc. Roy. Soc. Med. **29**:1295 (Aug.) 1936.

The paper is in two parts. In the first part Addison describes his methods of examination. In the second part he gives descriptions of the congenital abnormalities and other common urologic conditions. The cystoscopes used are: (1) a simple exploring cystoscope, no. 11 Charrière, which can be used for boys aged from 6 to 9 months; (2) a single catheterizing instrument of the same size; (3) a double catheterizing cystoscope, no. 14½ Charrière, which can generally be used for boys from 3 to 3½ years. He says that he has not been successful in cystoscopic instrumentation. For ureteric catheterization he uses the smallest catheter made (no. 9).

For taking pyelograms he uses a 13 per cent solution of sodium iodide. For intravenous pyelography he uses neo-iopax, giving doses of 8 cc. to infants and 12 cc. to older children. For testing renal efficiency he generally uses tests for the blood urea content and urea concentration tests.

The greater number of urologic diseases of children, he says, in part 2, are concerned with the anomalies and abnormalities of development of the urinary tract. The predisposing cause of pyuria is stagnation of urine, whether due to mechanical obstruction of the ureters, bladder or urethra by congenital atresia or caused by a developmental defect in their neuro-muscular mechanisms. He then describes the various abnormalities and anomalies he has seen in examining children in the Great Ormond Street Hospital for Sick Children and in the Lord Mayor Treloar Hospital at Alton. WILLIAMSON, New Orleans.

LIPOID NEPHROSIS AND AZOTEMIA. P. GAUTIER, Arch. de méd. d. enf. **39**:329 (June) 1936.

Clinical histories of three cases of lipoid nephrosis reported by Gautier in collaboration with Schoenau in 1933 are concluded and are compared with observations made by Pasteur Vallery-Radot, Delafontaine and Trombert in a more recent contribution on the evolution of lipoid nephrosis toward azotemia. Shortly after the first report one of the patients, a child, succumbed to a staphylococcic infection. Another child of 7 years, who presented the syndrome of lipoid nephrosis at the age of 3½ years, presented azotemia three years later and finally died in uremic coma. Autopsy in this case showed the characteristic lesions of chronic nephritis and birefringent lipoidosis.

The third report concerns a boy of 14. Four years previously he was admitted with a diagnosis of primary lipoid nephrosis, and in a few months this condition was complicated with azotemia. Marked improvement followed intercurrent otitis media, and complete arrest of all symptoms succeeded operation for mastoiditis. The conclusion is reached that azotemia may appear at any time in the course of this type of nephrosis but is more apt to be a dangerous complication when it appears late. AMESSE, Denver.

PURE LIPOID NEPHROSIS IN A GIRL TWO YEARS OLD. P. NOBÉCOURT, S. B. BRISKAS and TILIKICHEFF, Bull. Soc. de pédiat. de Paris **33**:330 (May) 1935.

A girl 2 years old was admitted to the hospital with marked edema as well as fluid in the serous cavities. Tests of the blood and urine indicated lipoid nephrosis. On a diet high in protein and with the administration of thyroid she was much improved three and a half months after the onset of her illness. However, this is regarded as probably only a remission in the course of the disease. During an infection of the respiratory tract there was a temporary increase in the nitrogen content of the blood. BENJAMIN, Montreal, Canada.

CORTICAL ABSCESSES OF THE RIGHT KIDNEY: DECAPSULATION FOLLOWED BY RECOVERY. JACQUES LEVEUF and HENRI GODARD, Bull. Soc. de pédiat. de Paris **33**:611 (Dec.) 1935.

A girl 13 years of age had an acute illness which was thought to be septicemia. One blood culture was negative. Pain developed in the region of the right kidney. The kidney was enlarged. The urine showed colon bacilli, a few white blood cells and no pus. Cystoscopic examination revealed deficient drainage on the right side, and the urine from that side contained colon bacilli but no pus. Roentgenographic examination after the intravenous injection of iopax showed incomplete filling of the calices and pelvis on the right side. An operation was performed, and the right kidney was found to be swollen and the capsule edematous. The latter was incised and stripped away from the kidney, which was studded with miliary cortical abscesses. The right ureter was malformed. The girl recovered. Examination later showed slight right renal insufficiency and dilated calices and pelvis with narrowing of the ureter on the right side. Decapsulation is considered the proper procedure when miliary cortical renal abscesses are present; if the condition of the patient does not improve, a secondary nephrectomy may later be necessary. BENJAMIN, Montreal, Canada.

HYDATID CYST OF THE KIDNEY IN A CHILD TEN YEARS OLD. PAUL GIRAUD, SALMON and JOUVE, Bull. Soc. de pédiat. de Paris **33**:718 (Dec.) 1935.

A 10 year old girl had an enlarged left kidney. There was a diagnosis of malignant tumor. At operation a hydatid cyst was found. This was excised, and the child recovered. She showed no eosinophilia, and Casoni's intradermal reaction with hydatid antigen was negative. How the parasite reached the kidney to produce a solitary renal cyst is difficult to explain.

BENJAMIN, Montreal, Canada.

THE CASE OF A FIFTEEN YEAR OLD GIRL WITH CHRONIC NEPHRITIS ASSOCIATED WITH ARTERIAL HYPERTENSION AND AZOTEMIA. P. NOBÉCOURT, Progrès méd., Aug. 22, 1936, p. 1327.

In addition to the report of the case described in the title, other cases of chronic nephritis in children are discussed.

LESLIE, Evanston, Ill.

CONGENITAL UNILATERAL KIDNEY. R. ERCOLE and A. FORT, An. de cir. **2**:384 (Nov.) 1936.

The case of a 41 year old man who presumably had congenital unilateral kidney is reported. The chief complaints were hematuria and loss of weight. On examination a freely movable large nontender kidney was felt. The urine was normal except for red blood cells. An intravenous pyelogram showed only one kidney, and that with a large, distorted upper pole. Cystoscopy revealed only one ureteral orifice, and the assumption was that there was congenital absence of the contralateral kidney. The extant kidney was thought to be involved by a neoplasm, but the patient's wish to leave the hospital prevented further investigation.

The authors review the literature on the subject. Congenital absence of one kidney is frequently associated with other anomalies of the genito-urinary tract. The necessity of distinguishing this condition from unilateral aplasia or atrophy of the kidney on one side is stressed. The paper is closed with a warning about the obvious danger of nephrectomy when there is only one kidney.

DIETRICH, Los Angeles.

PYONEPHROSIS IN AN INFANT. VÍCTOR SANTAMARINA and ANTONIO GUERNICA, Arch. de med. inf. **5**:298 (Aug.) 1936.

The condition in a 6 month old girl was double bilateral ureters and megalo-ureter, with termination in the head of the bladder. There was also gangrene of the sigmoid loop, but there were no symptoms of this before death. Photographs of the specimens are shown.

SANFORD, Chicago.

ECTOPIA OF THE TESTICLES. S. NETTO, Arch. de pediat., Rio de Janeiro **7**:412 (June) 1936.

There is no need for controversy as between surgical and medical intervention for undescended testes. In certain cases the abnormality will respond to gonadotropic substance and in others it will call for surgical treatment. Each method has its own field.

SANFORD, Chicago.

OVARIAN HORMONE IN THE TREATMENT OF GONORRHEAL VAGINITIS IN CHILDREN. F. ESCARDÓ and J. SALZMAN, Rev. méd. latino-am. **21**:1014 (June) 1936.

Escardó and Salzman describe the work of Lewis on gonococcic vaginitis in which the vaginal mucosa undergoes changes, assuming the histologic characteristics of the adult mucosa, following the injection of estrogen. They review the cases of Lewis, Brown, Reading, Huberman and Israeloff and report eight of their own in which good results were secured with an estrogenic substance. This method of treatment is promising, although still in the experimental stage.

DWYER, Kansas City, Mo.

A Case of True Hermaphroditism. S. Lindvall and F. Wahlgren, Virchows Arch. f. path. Anat. **297**:1, 1936.

A 19 year old person with predominately male anatomic and psychic characters had a small hypospadic penis and a descended small right testis. There was no beard, the breasts were like those of a young woman, and the pelvis was of female type. Regularly at monthly intervals the urine became bloody. At operation a uterus with a left tube and ovary were removed. The ovary was the size of that of a sexually mature young woman and was functioning actively. A portion of testis removed for biopsy revealed normal seminal tubules but no spermatogenesis; there were few interstitial cells. The authors consider the condition to be a gynandromorphism, in which the fertilized ovum is bisexual in its chromosomal constitution, rather than one of intersex, in which the developing embryo has the chromosomal constitution of one sex but the sexual characters later undergo alteration to those of the opposite sex. In the older classification of Klebs the anomaly would be true lateral hermaphroditism. Medicolegally it would have to be considered an example of sexus anceps, in which the sex cannot be legally determined.

Schultz, Evanston, Ill. [Arch. Path.]

Congo Red in the Diagnosis of Amyloid Disease of the Kidneys. C. I. Enesco and C. Isac, Bull. Acad. de méd. de Roumanie **1**:218, 1936.

Ten cubic centimeters of 1 per cent congo red was injected intravenously, and in one hour the blood serum and the urine were examined. In a normal person most of the dye is still in the blood at this time; the remainder has been excreted in the urine. In lipoid nephrosis the dye is rapidly passed through the kidneys and can be recovered in the urine. Amyloid disease of the kidneys apparently causes fixation of the dye in the affected organs, and after one hour congo red has disappeared from the blood and cannot be found in the urine.

Dietrich, Los Angeles.

Congenital Solitary Kidney. H. E. Shih and G. Y. Char, Chinese M. J. **50**:821 (June) 1936.

At the Peiping Union Medical College Hospital there has been no observation of congenital solitary kidney in 1,900 autopsies. The authors report two cases observed in the urologic service in the past fifteen years. These cases occurred in young men, neither of whom had any symptoms until the ages of 19 and 27 years, respectively. The authors discuss the symptoms which first suggest that something may be wrong with the kidneys. In the first case there had been for two years symptoms of increased frequency of urination and difficulty in starting the stream. In the other case there suddenly developed chill and high fever about a month prior to the patient's admission to the hospital, at which time pus cells were found in the urine. Excretory urography was used in both cases, a diagnostic measure by which congenital solitary kidney becomes recognizable. Of further value in such cases are examinations with the cystoscope and ureteroscope and roentgenography. The risk of operation on a solitary kidney requires the exercise of conservative judgment.

Neff, Kansas City, Mo.

Essential Fructosuria. J. van Lookeren Campagne, Maandschr. v. kindergeneesk. **5**:335 (May) 1936.

This is a description of a case of essential fructosuria in a boy of 6½ years, with determination of the dextrose and fructose content of the blood after ingestion of these sugars. The pathogenesis of this rare and remarkable disturbance has not yet been elucidated. In the author's opinion the prognosis is favorable, but he recommends omitting fructose and saccharose from the food.

van Creveld, Amsterdam, Netherlands.

Diseases of the Ductless Glands; Endocrinology

THYMOMA AND THYMIC HYPERPLASIA IN MYASTHENIA GRAVIS. E. H. NORRIS, Am. J. Cancer **27**:421, 1936.

Four cases of myasthenia gravis with autopsies are reported. In two of these a high degree of hyperplasia of the thymus was observed; in the other two there was no gross evidence of thymic involvement. In fifty per cent of the reported cases of myasthenia gravis in which autopsies were performed there was some form of thymic involvement. Pathologic conditions of the thymus which have been regarded as benign tumors have been found in half of the cases of myasthenia gravis in which demonstrable changes in the thymus were recognized. Such conditions Norris has interpreted as conditions of extreme epithelial hyperplasia. Pathologic conditions which have been regarded as enlargement or persistence of the thymus have been found in half of the cases of myasthenia gravis in which demonstrable changes in the thymus were recognized. These he has interpreted as conditions of moderate epithelial hyperplasia.

FROM THE AUTHOR'S SUMMARY. [ARCH. PATH.]

JUVENILE DIABETES AND TUBERCULOSIS. A. L. NEWCOMB, Am. Rev. Tuberc. **32**:507 (Nov.) 1935.

Eight children of a group of fifty with juvenile diabetes reacted positively to the tuberculin test. Definite calcification was found in the lungs of one child, and in another there was a questionable calcium deposit with large hilar lymph nodes. One boy who gave a negative reaction to the test on admission gave a violent positive reaction four months later, and roentgenograms revealed a fairly wide area of increased density about the hilus of each lung. His mother was found to have active pulmonary tuberculosis. No evidence of phthisis has been found in any roentgenograms of the chest up to the present.

EUGENE SMITH, Ogden, Utah.

NEUROENDOCRINE APLASIA AND POLYDACTYLISM OF THE LAURENCE-MOON-BIEDL SYNDROME. SAMUEL WEINGROW, Arch. Pediat. **53**:564 (Aug.) 1936.

Weingrow reports a case of a condition occurring in a white boy 5 years old which he considers to be the Laurence-Moon-Biedl syndrome.

The main features are cerebral aplasia characterized by cortical atrophy, hydrocephalus, hemiparesis, jacksonian convulsions, nystagmus and increased intracranial pressure resulting in pallor bitemporally. Associated with the neural aplasia there were aplasia of one testicle and polydactylism. No mention is made of the child's mental age. The author contends that the underlying cause of this disease is aplasia of the cerebrum, and not a lesion of the hypothalamus, as Laurence, Moon and Biedl believed.

ORR, Buffalo.

ACUTE ABDOMINAL PAIN IN JUVENILE DIABETES MELLITUS. ALVAH L. NEWCOMB, Illinois M. J. **68**:544 (Dec.) 1935.

In a group of seventy diabetic children there were two with abdominal pain due to ketosis, four with acute appendicitis, one of whom also had ketosis, and one with a fecolith who was thought to have appendicitis.

Newcomb believes that acute appendicitis is to be differentiated from diabetic ketosis with abdominal pain mainly by a history of localized pain, associated with localized rigidity and tenderness and moderate fever. When the history of pain is less definite and there is more vomiting and drowsiness with generalized tenderness, diabetic ketosis should be ruled out by proper diabetic therapy.

BARBOUR, Peoria, Ill.

REPORT OF A CASE OF SIMMONDS' DISEASE. CHARLES W. DUNN, J. Nerv. & Ment. Dis. **83**:166 (Feb.) 1936.

A 12 year old girl was seized suddenly with the symptoms frontal headache, extreme nervousness, loss in weight, cessation of the menses and severe, rapidly progressing general weakness. Her previous history was irrelevant. On examination she appeared emaciated and her pulse was barely perceptible. She was extremely pale and was subject to vertigo. Her breasts were atrophied, and her external genitalia were smaller than normal for her age. Her hair was lusterless and her nails pitted. Laboratory tests revealed nothing pertinent except that her basal metabolic rate was minus 3.

A roentgenogram of her chest showed no pulmonary tuberculosis. A roentgenogram of the lateral aspect of her right elbow showed complete bony union of the center of ossification from the olecranon to the shaft, with apparent disappearance of the epiphysial line. A lateral view of her right ankle showed evidence of complete union of the center of ossification, the os calcis and its posterior extremity. The sphenoic angle was 155 degrees (more acute than is normal for a girl of 12).

Therapy consisted of a daily injection of 2 cc. of solution of anterior pituitary (Armour) and a weekly injection of 10,000 rat units of an estrogenic substance. At the end of three months of this treatment definite evidence of improvement was noted. The improvement remained, and there was a gain in weight of 26 pounds (11.8 Kg.) in five months. The intervals between injections were gradually being lengthened, and the injections were to be discontinued when normalcy was attained.

Dunn gives a comprehensive discussion of this interesting disease.

JAHR, Omaha.

THE THYMUS AND ADRENALS IN THE RESPONSE OF THE ORGANISM TO INJURIES AND INTOXICATIONS. H. SELYE, Brit. J. Exper. Path. **17**:234, 1936.

In the rat a syndrome consisting of rapid involution of the thymus, formation of a pleural transudate and enlargement of the adrenals with loss of cortical lipoids is produced by certain operative injuries, drugs, low temperatures and stimuli causing merely nervous excitement. In fact, among all the stimuli tried, none has been found which would seriously damage the experimental animal without causing rapid involution of the thymus and enlargement of the adrenals. The involution produced by such means is more pronounced than that which follows withdrawal of all food and water, and therefore it is not due to failure of the animal to eat. All the injuries and drugs which normally cause involution of the thymus are without effect on the size of the thymus of an adrenalectomized animal. In the hypophysectomized rat they have some effect but much less than in the normal rat. A method for removing the adrenal medulla with a suction pump is described. This method allows removal of the medulla without serious injury to the cortex. The thymus of a rat thus deprived of medullary tissue but still in possession of active cortical tissue shows little if any ability to involute. The inability of the thymus of the adrenalectomized animal to undergo involution is not restored by sodium chloride, extract of adrenal cortex in such doses as have been used, or epinephrine. Sodium chloride heightens the otherwise low resistance of adrenalectomized rats to toxic doses of various drugs if the animals receive food. It is inactive, however, in fasting animals. The changes caused by a drug when it is given for the first time will subside later in spite of the continued administration of the drug. It appears, therefore, that the syndrome described, consisting of thymic edema followed by involution, formation of a pleural transudate, enlargement of the adrenal cortex with loss of its lipoid granules and rapid loss of body weight, represents an "alarm reaction" which enables the organism to meet critical situations more efficiently. Attention is called to the possible connections between these experimental findings and the clinical picture of drug habituation, on the one hand, and nonspecific fever and protein therapy, on the other.

FROM THE AUTHOR'S SUMMARY. [ARCH. PATH.]

THE TREATMENT OF SEVERE DIABETES IN CHILDREN WITH PROTAMINE INSULINATE. T. IZOD BENNETT and A. MORTON GILL, Lancet **2**:416 (Aug. 22) 1936.

Four children suffering from severe diabetes were treated with protamine insulinate. With this method the total daily dose could be reduced; the patients were kept free from sugar; only two daily doses were necessary, and all severe reactions were abolished. All the patients asserted that their general well-being was greatly increased.

LANGMANN, New York.

SURGICAL TREATMENT OF A CASE OF EXOPHTHALMIC GOITER IN A CHILD. J. HUBER, PETIT-DUTAILLIS and SUZANNE DREYFUS, Bull. Soc. de pédiat. de Paris **33**:336 (May) 1935.

A boy 13 years old who had exophthalmic goiter was treated surgically. Local anesthesia was used, and the left lobe of the thyroid was removed. Five months later the child was markedly relieved of his symptoms, although his basal metabolic rate was still plus 34, and exophthalmos was present. The cholesterol content of the blood at this time was normal. Because of the importance of the thyroid in the growth and development of the child, less of the gland should be excised than in the adult with exophthalmic goiter, as it is better to leave a little more of the gland than is necessary even though some evidence of hyperthyroidism may remain, such as a high basal metabolic rate, than to produce myxedema by removing too much. With the administration of small doses of iodine the basal metabolism usually returns to normal.

BENJAMIN, Montreal, Canada.

HYPERTROPHY OF THE THYMUS AND SUDDEN DEATH IN AN INFANT FOUR MONTHS OLD. PAUL GIRAUD, SALMON and JOUVE, Bull. Soc. de pédiat. de Paris **33**:724 (Dec.) 1935.

A girl 4 months of age was admitted to the hospital for operation on a bilateral harelip. She had increased substernal dulness to the left and right, which corresponded to a widened mediastinal shadow seen in a roentgenogram of the chest. Operation was postponed. Pharyngitis developed and the infant died suddenly. Necropsy confirmed the presence of a large thymus, which weighed 40 Gm.

BENJAMIN, Montreal, Canada.

SERUM LIPASE CONTENT IN MYXEDEMA. F. WENDEL, Monatschr. f. Kinderh. **66**:288 (Aug.) 1936.

In myxedema the lipase content of the blood is higher than normal. In rabbits the administration of thyroid extract leads to a decreased content of lipase. Wendel found no such effect, however, in myxedematous or normal children.

GERSTLEY, Chicago.

Diseases of the Muscles, Bones, Joints and Lymph Glands

A PROBABLE ETIOLOGIC FACTOR IN INFANTILE ECZEMA BASED ON X-RAY EXAMINATIONS OF THE GASTRO-INTESTINAL TRACT. A. STRICKLER, M. K. FISHER and H. LOWENBURG, Arch. Pediat. **53**:397 (June) 1936.

The authors advance the theory that in some cases infantile eczema is due to gastric retention or intestinal stasis. Roentgenologic studies were made on twenty-eight children with infantile eczema. Their ages ranged from 9 months to 11 years. Forty-three per cent of the series had evidence of gastric retention; 53 per cent showed intestinal stasis; 29 per cent had both gastric and intestinal stasis. No mention is made of the therapeutic results.

ORR, Buffalo.

THE DIAGNOSIS OF MESENTERIC LYMPHADENITIS. E. P. COLEMAN, Illinois M. J. **68**:408 (Nov.) 1935.

Acute mesenteric lymphadenitis is being found with an increasing frequency in patients with acute abdominal symptoms.

In a review of eighty-six cases Coleman found that an infection of the upper respiratory tract or of the gastro-intestinal tract preceded each attack. Most of the patients were children or young adults.

He found colicky pain to be the one constant symptom. Nausea and vomiting were more common in older patients. The temperature usually ranged from 100 to 101 F. The leukocyte count averaged 11,000. Muscular rigidity was not usually seen. Abdominal tenderness was more generalized than it is in acute appendicitis. The condition is difficult to distinguish from acute appendicitis.

BARBOUR, Peoria, Ill.

THE HEREDITARY ASPECT OF PROGRESSIVE PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY. G. DEN. HOUGH JR., New England J. Med. **214**:1189 (June 11) 1936.

A family history presenting six cases of the disease, one of which is questionable, is reported. Seventy-nine known members are recorded. In all cases the transmission in this family was through an apparently normal female.

Hough believes that there is increasing evidence to substantiate Barker's statement that the facioscapulohumeral, or Landouzy-Dejerine, type of progressive muscular dystrophy is transmitted as a dominant characteristic. As the patients frequently live to sexual maturity, they should be warned against having offspring. The more common pseudohypertrophic, or Duchenne, type of progressive muscular dystrophy is apparently transmitted through the clinically normal female members of the family, exactly as is hemophilia. Perhaps this is due to the fact that the person with disease of this type is almost always incapacitated before sexual maturity. There is sufficient evidence to prohibit reproduction by the apparently normal females in such a family, while permitting offspring to the apparently normal males. The method of inheritance may be used as a basis of sound classification, as it is directly concerned with the prevention of the condition.

GENGENBACH, Denver.

BACTERIOLOGIC STUDIES IN MYASTHENIA GRAVIS. E. C. ROSENOW and F. R. HEILMAN, Proc. Soc. Exper. Biol. & Med. **34**:419 (May) 1936.

Streptococci have been isolated from the nasopharynx and other foci of infection and sometimes from the muscles of patients with myasthenia gravis. These bacteria resemble morphologically and culturally the green-producing streptococci isolated from similar sources in diseases other than those affecting nerve and muscle, but they differ markedly in cataphoretic velocity and specific virulence. In monkeys and rabbits injections of living and heat-killed streptococci isolated from the tissues of persons with myasthenia gravis produced progressive weakness, undue fatigue on exertion and characteristic microscopic lesions. The organisms were isolated from the muscles of the animals which exhibited these myasthenia gravis-like symptoms. Control animals given injections of streptococci causing diseases not involving muscle showed no myasthenia-like symptoms, nor were the lesions in the muscle found. Patients with myasthenia gravis were found to be extremely sensitive to therapeutic subcutaneous injections of the heat-killed streptococci and filtrates.

HANSEN, Minneapolis.

SEROLOGIC STUDIES WITH STREPTOCOCCI ISOLATED IN CASES OF MYASTHENIA GRAVIS. E. C. ROSENOW and F. R. HEILMAN, Proc. Soc. Exper. Biol. & Med. **34**:477 (May) 1936.

Patients with myasthenia gravis harbor a streptococcus of specific serologic type. They have antibodies in their blood specific for this organism, and they have in their tissues an antigen of this type of streptococcus.

HANSEN, Minneapolis.

ACUTE OSTEOMYELITIS: ETIOLOGY, SYMPTOMS, DIAGNOSIS AND TREATMENT OF EIGHTY CASES. J. I. MITCHELL, South. M. J. **29**:588 (June) 1936.

Acute osteomyelitis is more prevalent during the period of rapid osseous growth. Males are affected more frequently than females. The long bones are more often involved, the femur and tibia being most commonly affected. Trauma, infected cutaneous lesions, oral sepsis and debility from malnutrition or disease are predisposing causes. Staphylococcus aureus was the etiologic agent in 65 per cent of a series of eighty cases. The mortality was highest (33.3 per cent) in those cases in which the condition was caused by Streptococcus haemolyticus. There were six deaths in the group, a mortality rate of 7.5 per cent.

In about a third of the cases healing occurred following surgical drainage of pus during the acute stage of the disease.

SCHLUTZ, Chicago.

MORQUIO'S DISEASE: REPORT OF TWO CASES. P. SUMMERFELDT and ALAN BROWN, Arch. Dis. Childhood **11**:221 (Aug.) 1936.

A report is presented of two brothers with a generalized, symmetrical osseous disturbance resulting in dwarfism and a peculiarly shaped chest. Roentgenologically the chief changes appear in the vertebrae, pelvis and long bones, and there is delay in the development of the epiphyses, especially of the head of the femur. The condition has a tendency to be familial, and its etiology is unknown at present.

KELLY, Milwaukee.

TREATMENT OF ARTHRITIS BY HYDROTHERAPY. W. S. C. COPEMAN, Brit. J. Phys. Med. **11**:22 (June) 1936.

This is a review of the different types of hydrotherapy. The treatment of chorea by immersion for from one to one and a half hours in a bath of water at or just above the body temperature is believed to have reduced the percentage of relapses.

HARTMAN, Cleveland.

INTESTINAL OBSTRUCTION IN AN INFANT DUE TO AN UNUSUAL CAUSE. A. PERROT and L. DANON, Ann. d'anat. path. **12**:157, 1935.

In a 15 day old infant with symptoms of intestinal obstruction and fecal vomiting, operation disclosed no mechanical obstruction. At autopsy there was found marked hypertrophy of the small intestine, especially of the ileum (resembling Hirschsprung's disease of the colon). The hypertrophy was due entirely to a thickening of the muscular layer. The terminal portion of the ileum and the proximal portion of the colon were not hypertrophic but were found to have almost complete absence of the plexus of Auerbach. The authors believe that either paresis or spasm of this segment resulted in the intestinal obstruction, with secondary hypertrophy of the bowel above. They believe that this segment of the intestinal tract (terminal portion of the ileum and proximal portion of the colon) may have a separate innervation, so far not differentiated by the physiologists.

MELNICK, Decatur, Ill. [ARCH PATH.]

EMBRYONIC RETICULOSARCOMA OF BONE MARROW: EWING'S SARCOMA OF A GRAVE TYPE. ROBERT CLÉMENT and JEANNE DELON, Bull. Soc. de pédiat. de Paris **33**:649 (Dec.) 1935.

A boy 8 years old was taken ill with painful torticollis which prevented him from sleeping. He had no fever. Despite a negative reaction to the tuberculin test, it was thought that he was suffering from Pott's disease. Roentgenograms showed involvement of the bone marrow of practically all the bones. Biopsy revealed neoplastic tissue, which consisted of embryonic undifferentiated cells. There was marked anemia, but the blood smears showed no immature cells, even shortly before death. There was no Bence-Jones substance in the urine. The site of the primary malignant tumor was not determined.

BENJAMIN, Montreal, Canada.

CONGENITAL MYOTONIA OF THE WERDNIG-HOFFMANN TYPE. B. WEILL-HALLÉ, DELTHIL and RAMBERT, Bull. Soc. de pédiat. de Paris **33**:661 (Dec.) 1935.

A girl 3 months old presented the picture of amyotonia congenita. There was no family history of this disease. As a reaction of degeneration was obtained in almost all the muscles, the condition was thought to fall into the category described by Werdnig and Hoffmann. It is pointed out that clinically there is no sharp line of demarcation between this condition and Oppenheim's disease. The infant died suddenly with signs of bulbar involvement.

BENJAMIN, Montreal, Canada.

PERIOSTEAL DYSPLASIA: REPORT OF A CASE. G. DREYFUS-SÉE, Bull. Soc. de pédiat. de Paris **33**:679 (Dec.) 1935.

A boy 7 months old had multiple fractures with callus about many of them, due to osteogenesis imperfecta. The calcium contents of the blood, urine and stools were practically normal. Viosterol and calcium as well as thyroid and pituitary extracts were given, but no unusually rapid improvement followed.

BENJAMIN, Montreal, Canada.

A CASE OF HODGKIN'S DISEASE. E. CUNHA, J. de pediat., Rio de Janeiro **3**:243 (June) 1936.

A 4 year old Negro boy showed great generalized adenopathy. Autopsy disclosed the typical Paltauf-Sternberg cells, confirming the diagnosis.

SANFORD, Chicago.

SECONDARY ZONULAR OPACITY OF THE CORNEA IN POLYARTHRITIS CHRONICA LEUCOCYTOTICA (STILL) AND LYMPHOCYTOTICA (RHONHEIMER). J. KARSCH, Arch. f. Augenh. **110**:106, 1936.

Polyarthritis chronica leucocytotica, or Still's disease, is an infectious disease of childhood of unknown etiology. The signs are fever, enlargement of the spleen and the lymph glands and inflammation of the small joints in the form of periarthritis. The bones and the cartilage often remain normal for years. The fluid in the cavities of the joints contains polymorphonuclear leukocytes. The disease usually begins at about the age of 8 and may last for years. Adhesive pericarditis frequently develops.

The form described by Rhonheimer differs little from this form, with the exception that the fluid aspirated from the joints contains lymphocytes.

Karsch reports two cases of this disease, in both of which there were an old iridocyclitis and zonular opacity of the cornea. Syphilis and tuberculosis could be excluded from the diagnosis. He considers the involvement of the cornea and iris as part of the picture of the disease.

ADLER, Philadelphia. [ARCH. OPHTH.]

THE ETIOLOGY AND PATHOGENESIS OF SOLITARY CYSTS OF BONE. W. WEBER, Fortschr. a. d. Geb. d. Röntgenstrahlen **53**:861 (June) 1936.

This is an excellent review of the subject indicated in the title. It is amplified by a report of histologic examinations in five cases of solitary cyst in persons whose skeletons were otherwise normal. Trauma does not appear to be the fundamental cause of the solitary cyst. The author's investigations support the point of view first expressed by von Mikulicz and recently reaffirmed by Geschickter and Copeland, that the solitary cyst of bone is the result of a developmental disturbance in the growth of bone. The bibliography is comprehensive, and the illustrations are numerous.

MCCUNE, New York.

A Rare Deformity of the Knees. H. Scheuermann, Röntgenpraxis **8**:364 (June) 1936.

At the age of 14 years a youth commenced to exhibit progressive deformity of both knee joints, which was shown roentgenographically to be the result of anterior displacement of the proximal epiphyses of the tibias. The lesion became stationary at the end of four years. The patient also had juvenile kyphosis (Scheuermann). The cause of the developmental anomaly of the knees was not clear; presumably trauma acted as a precipitating factor.

McCune, New York.

Incomplete "Block-Vertebra" of the Upper Thoracic Spine. J. E. W. Brocher, Röntgenpraxis **8**:380 (June) 1936.

Kyphosis is commonly acquired, due to spondylitis or to trauma; however, it may be congenital. The latter possibility is exemplified in the case of an 11 year old girl who presented a curvature of the spine caused by hypoplasia of the body of the third thoracic vertebra with fusion of the spinous processes of second and third thoracic vertebrae and narrowing of the intervertebral disk between these two structures. The lesion was nonprogressive

McCune, New York.

Congenital Muscular Hypertrophy of an Upper Extremity. H. von Brücke, Virchows Arch. f. path. Anat. **296**:681, 1936.

A boy born one month prematurely presented no external maldevelopments except enlargement of the left upper extremity. The increase in size involved chiefly the forearm and the hand. The circumference of the left arm was 9.5 cm., as compared with 8.8 cm. for the right arm; the left forearm was 11.2 cm. in circumference and the right 8.6 cm.; the left hand measured 11 cm. in circumference and the right 7.7 cm. The upper extremities were approximately equal in length; the bones were equal in length, but those of the left arm were somewhat thicker. The increase in the size of the hand appeared to be due largely to subcutaneous fat, but that of the forearm and arm, to muscular hypertrophy. The muscular strength on the enlarged left side was greater at birth and at an examination three months later. Von Brücke places the anomaly in the group termed "sector maldevelopments," in which the malformed portion is usually of less functional value than normal. In the present instance, the greater muscular strength is indicative of an excessive formation of normally functioning tissue. The possible later development of evidences of neurofibromatosis is considered.

Schultz, Evanston, Ill. [Arch. Path.]

Diseases of the Eye

Virus of Inclusion Conjunctivitis: Further Observations. P. Thygeson and W. F. Mengert, Arch. Ophth. **15**:377 (March) 1936.

The authors have confirmed the virus etiology of the nonbacterial conjunctivitis of the new-born. Evidence is presented to indicate that the basophilic cytoplasmic inclusion bodies are virus colonies similar to those of psittacosis. Evidence derived from experiments as to transmission seemed to demonstrate the etiologic identity of the inclusion blennorrhea and the nontrachomatous inclusion diseases of adults.

The authors report eight additional cases of inclusion blennorrhea in infants, making a total of nineteen cases reported by them. Sixteen of the eighteen mothers in whose infants inclusion blennorrhea developed presented no clinical signs of genito-urinary infection. Microscopic examination revealed the presence of inclusion bodies in epithelial cells from the cervix in seven of ten cases, and secretions from the vagina in four instances induced follicular conjunctivitis, which is characteristic of infection due to inclusion virus, in baboons.

Wescott, Chicago.

ROENTGENOGRAPHIC DIAGNOSIS OF RETINOBLASTOMA. RAYMOND L. PFEIFFER, Arch. Ophth. **15**:811 (May) 1936.

Because calcareous degeneration takes place in retinoblastoma (glioma), roentgenographic evidence of opacity was sought as an aid in diagnosing this ocular tumor. Roentgenograms of twenty enucleated eyes with retinoblastoma revealed calcium deposits in fifteen. In fourteen cases in which the eyes were in situ when retinoblastoma was diagnosed, roentgenograms were made before operation. In ten of these fourteen cases glioma was demonstrated by histologic examination. In eight of these ten cases typical shadows were obtained. In two cases calcium was not found before enucleation or later by roentgen or histologic examination.

WESCOTT, Chicago.

PIGMENTED DEPOSITS OF THE LENS AND CORNEA OF DOUBTFUL NATURE. R. E. WRIGHT and K. K. NAYAR, Brit. J. Ophth. **20**:295 (May) 1936.

A Hindu youth aged 17 was seen on Dec. 19, 1924, because of inflammation of the conjunctiva. The condition was of a mixed trachomatous and eczematous nature. There was pannus, more marked above. He had been treated for some time with silver nitrate, copper sulfate, mild protein silver and zinc. In March 1928 the conjunctival and corneal condition was aggravated, and there was a "powdery fluorescent layer just under the lens capsule." In 1929 he was seized with what was probably an attack of malaria. The local treatment included the use of the thermophore and radium. In January 1930 staining of the cornea was definite.

At present there are the remains of a generalized pannus. The corneas present a fairly uniform olivaceous tint. On the anterior capsule are rounded yellowish-brown opacities, 3 mm. in diameter, with chocolate-brown, somewhat sinuous borders, surrounded by a narrow zone of fine haze. The condition is almost symmetrical in the two eyes.

A striking feature was a shadow of occluded superficial vessels thrown on a gray-green, opaque-looking background apparently coincident with Descemet's membrane. The lens otherwise appeared clear. The pigment deposit seemed to be in the endothelium. With high power the anterior capsular opacity looked like a fine metallic deposit the color of old gold. Surrounding it like a corona were fine radiating whitish lines in the superficial layers of the lens.

Wright and Nayar consider the condition to have been produced either by the metallic compounds used in treatment or by broken-down hemoglobin. They believe there was ample opportunity for local disintegration of hemoglobin.

ZENTMAYER, Philadelphia. [ARCH. OPHTH.]

LYMPHANGIOMA IN THE ORBIT. NORMAN M. DOTT, Tr. Med.-Chir. Soc. Edinburgh, 1936, p. 76; in Edinburgh M. J., June 1936.

A child 10 years old at the time of presentation acquired proptosis of one eye at the age of 2 years; at 5 marked protrusion of the eye was accompanied by local edema and severe conjunctivitis. The biopsy showed a primitive lymphangiomatous tumor, with numerous developing lymph follicles. The tumor was successfully treated by roentgen rays and the eye receded to the normal position, but because of scarring, shrinking and discomfort it had to be removed finally.

NEFF, Kansas City, Mo.

A CLINICAL AND ELECTRICAL STUDY OF HEREDITARY AND FAMILIAL PARTIAL FACIAL DIPLEGIA. R. HUMBERT and R. ROSSANO, Ann. d'ocul. **173**:369 (May) 1936.

Facial diplegia of cortical origin is a syndrome well known in France, particularly since the work of Charles Foix and his pupils. It has interested ophthalmologists from more than one point of view. As the name indicates, it

affects not only the muscles of the face, particularly the orbicular muscles of the eyelids, but frequently the oculomotor muscles, so that there are disturbances of sight.

Acquired facial diplegia of peripheral origin begins as a lesion of the peripheral neuron of the facial nerve and is more evident on one side than on the other. One would think that this condition is exceptional, but it has been noted by several authors. The cases are, however, extremely rare. Recently, Alajouanine, Huc and Gopcevitch reported the occurrence, in four children, of a congenital syndrome characterized by double clubfoot, double facial paralysis and double paralysis of the sixth nerve. Humbert and Rossano have recently observed in a young man who wished to be operated on for bilateral ptosis an incomplete facial diplegia, which he asserted was not only congenital but also familial and hereditary.

The details of the case, two diagrams and a bibliography are given.

McKee, Montreal, Canada. [Arch. Ophth.]

Hereditary Luxation of the Lens and the Syndrome of Marfan. Delord and Viallefont, Bull. Soc. d'opht. de Paris, January 1936, p. 44.

A father, aged 41, and his son, aged 15, showed various stigmas of the syndrome of Marfan. Both had bilateral luxation of the lens. Scoliosis, arachnodactylia, hyperactive reflexes, absence of secondary sexual characteristics, infantile testicles, a basal metabolism lowered 25 per cent and a blood calcium content of 13 mg. per hundred cubic centimeters were found for the son, but the father was normal in all these respects. Delord and Viallefont discuss the various theories of the etiology of the condition. Excellent photographs of the son are shown in the article.

Mayer, Chicago. [Arch. Ophth.]

Congenital Pigmented Cysts of the Iris and the Ciliary Zone; Vestige of the Vascular Membrane of the Lens; Persistence of the Hyaloid Artery. F. Badeaux, Bull. Soc. d'opht. de Paris, January 1936, p. 68.

The clinical rarity of congenital pigmented cysts of the iris and the ciliary zone and the infrequency of reports of the condition as studied by means of the slit lamp or by pathologic examination prompted this article. In a patient 10 years of age, on dilation of the left pupil a mass was found back of the iris between 6 and 9 o'clock. Pictures taken with the use of the slit lamp are reproduced. The history of such cysts is reviewed, and drawings are shown to demonstrate their embryologic development. Badeaux believes that they are caused by doubling of the epithelium of the uveal layer and subsequent incomplete absorption. Surgical intervention is indicated if the visual acuity is lowered. If such a growth is watched over a long period its benign nature is confirmed.

Mayer, Chicago. [Arch. Ophth.]

A Case of Convergence Spasm. P. Bailliart, Bull. Soc. d'opht. de Paris, February 1936, p. 81.

A girl aged 8 years had complained of double vision for eight days. The mother reported that the child had had such an attack twice previously, with accompanying convergent strabismus and apparent diplopia. The first attack was at the age of 2 years and lasted eight days; the second, which occurred after a year, was of short duration. These two attacks were thought to be due to eating chocolate. In the present attack the strabismus was 40 degrees. A lens to correct a refractive error of 1 diopter brought the vision of the right eye to 10/10 and that of the left to 9/10. When the child put on her glasses (a + 1.0 diopter sphere) the convergence disappeared. Also, if her attention was distracted or she became greatly interested in something no evidence of crossing was noted. Bailliart injected a 2 per cent solution of procaine hydrochloride into the right internal rectus muscle; this caused complete disappearance of the squint. Other

facts of the history were unimportant. Physical examination, neurologic examination and examination of both parents gave entirely negative results. Bailliart thinks that the condition was spasm of convergence, of which there are few reports in the literature. In some cases the condition was noted after encephalitis. No etiologic factor was found in the described case.

MAYER, Chicago. [ARCH. OPHTH.]

CHANGES IN THE IRIS CAUSED BY SYPHILIS, WITH CONGENITAL ECTOPIA LENTIS. O. KURZ, Arch. f. Augenh. **109**:592, 1936.

Kurz reports a typical case of arachnodactylia associated with congenital dislocation of the lens. The patient had acquired syphilis, and parenchymatous keratitis developed with iritis. The latter resulted in depigmentation of the pupillary margin of the iris. Kurz explains this as due to the neurotrophic activity of the syphilitic virus in the secondary stages. He regards it as a local manifestation of the general debility of the sympathetic nervous system in this disease.

ADLER, New York. [ARCH. OPHTH.]

OCCUPATIONAL ABILITY OF PERSONS OPERATED ON FOR CONGENITAL CATARACT. K. VOM HOFE, Arch. f. Augenh. **110**:35, 1936.

Of twenty-one patients with bilateral congenital cataract who had been operated on, ten had good visual acuity. Nine had sufficient to pursue their usual occupations. In only two instances was the visual acuity insufficient for the patient to learn a trade. In the vast majority vom Hofe found the visual acuity sufficient for the patient's employment as locksmith, carpenter, merchant, farmer, messenger, servant, student or clergyman.

ADLER, Philadelphia. [ARCH. OPHTH.]

MORPHOLOGY AND DEVELOPMENT OF ANOMALIES OCCURRING IN MICROPHTHALMOS. F. RINTELEN, Arch. f. Ophth. **135**:120 (Jan.) 1936.

In an eye which was removed from a new-born infant with the diagnosis of pseudoglioma a persistent hypertrophic membrana capsulohyaloidea with persistent hyaloid artery was found to be the anatomic substratum of the pseudoglioma. Besides there were other anomalies, viz., hyperplasia of the retina, posterior cortical and polar cataract, and formation, by misplaced capsular epithelium, of a rudimentary lens within the persistent hyaloid membrane.

KRONFELD, Chicago. [ARCH. OPHTH.]

ACID AND ALKALI BURNS OF THE EYE. D. I. BEREZINSKAYA, Sovet. vestnik oftal. **8**:319, 1936.

This is an experimental study of chemical burns of the eye. During thirty-five years (1900 to 1935) there were admitted to the hospital of Helmholtz 876 patients who suffered from ocular burns caused by chemical, thermic and explosive agents. Of these, 196 had acid and 229 alkali burns. On rabbits' eyes Berezinskaya experimented with a 50 per cent solution of sulfuric acid and a 20 per cent solution of sodium hydroxide, but destruction of the eyes followed too rapidly. She therefore used a 30 per cent solution of sulfuric acid, an 18 per cent solution of hydrochloric acid and a 5 per cent solution of sodium hydroxide; these concentrations are met with more frequently in industrial and chemical hazards.

In the first series of experiments (on seven eyes of four rabbits) a study was made of the rapidity of the penetration of alkali and acids into the anterior chamber and vitreous. Sodium hydroxide was observed in the anterior chamber within five minutes after its instillation into the conjunctival sac. It was found in the secondary aqueous within thirty minutes and within twenty-four hours. No alkali was observed in the vitreous at any time. Sulfuric acid did not enter the anterior chamber or the vitreous in five minutes, ten minutes or three hours.

In the second series seven eyes were burned with acids and seven with alkali. The clinical picture was studied with focal illumination and with the slit lamp. The eyes were enucleated at from four hours to twenty-two days after the burn and studied histologically. Detailed protocols of the experiments, five photomicrographs and two tables are given which illustrate the subject well. Some histories of patients in the hospital who had suffered burns with alkali and acids are given for comparison.

Berezinskaya comes to the following conclusions: 1. Alkali (5 per cent sodium hydroxide) penetrates the anterior chamber within five minutes. 2. Acid (30 per cent sulfuric acid) does not penetrate the anterior chamber. 3. Alkali and acid burns differ histologically. 4. The difference is noticeable in the first few hours, which explains the difference in the penetrability of the cornea for acids and alkali. 5. The pathologic process in alkali burns is diffuse. It affects all parts of the cornea except Descemet's membrane. The uveal tract is involved. The lens remains intact. 6. The process in acid burns is limited, superficial. Secondary infection aids the spreading of the process into the deep layers with defect of tissue and formation of crusts. The lens becomes cataractous. The uveal tract is not always involved. 7. Descemet's membrane is very stable to both alkali and acids. 8. Characteristic of hydrochloric acid burns are the homogeneity of the corneal tissue and crust formation on the surface. 9. Acid burns frequently cause cataracts. 10. Clinically and histologically, alkali burns are more severe than acid burns.

SITCHEVSKA, New York. [ARCH. OPHTH.]

SUCCESSFUL REMOVAL OF AN EYELASH FROM THE POSTERIOR CHAMBER. R. I. BRUFMAN, Sovet. vestnik oftal. **9**:70, 1936.

A boy aged 11 was seen two days after he was hit in the right eye by a wire. The eye was inflamed and showed a narrow strip of hypopyon. There was a perforating wound at the limbus between 5 and 6 o'clock from which an eyelash was protruding. The latter was removed easily with forceps. Three days later, when the exudate absorbed from the anterior chamber and the pupil was dilated, another eyelash was seen in the pupillary region, with its other end hidden behind the iris. A corneal incision was made with a keratome at about 6 o'clock. A toothless iris forceps was introduced into the anterior chamber, and the eyelash was pulled out without injury to the iris or the lens. The inflammation of the eye subsided in three days, and the vision improved from 0.3 to 0.7.

SITCHEVSKA, New York. [ARCH. OPHTH.]

Skin Diseases; Allergy

FAMILIAL XANTHOMA: REPORT OF THREE OF FIVE SIBLINGS WITH XANTHOMA TUBEROSUM MULTIPLEX. E. A. LEVIN and M. SULLIVAN, Arch. Dermat. & Syph. **33**:967 (June) 1936.

The authors report on three brothers, 12, 13 and 15 years of age, respectively, who had xanthoma tuberosum multiplex. Two other siblings were not afflicted, and no family history of xanthoma was obtained. Each of the patients had increases in the cholesterol, lecithin and total fat contents of the blood. Two of the boys had increases in the basophil count of the blood to 6 and 20 per cent, respectively. Biopsies in all three cases showed the typical histopathologic picture of xanthoma.

DULIN, Iowa City.

SPHACELODERMA: REPORT OF A CASE OF UNUSUAL PHAGEDENIC ULCERATION OF THE SKIN, SUBCUTANEOUS TISSUE AND MUSCLE OF THE CHEST WALL. W. SACHS, Arch. Dermat. & Syph. **33**:977 (June) 1936.

Sachs reports the case of a girl 10 years of age in whom there developed without apparent cause what appeared to be a furuncle of the left axilla. This

was opened, and several days later the lesion began to slough. A month later a superficial fluctuating area developed to the left of the sternum; this was drained, and a few days later it began to slough. The sloughing continued until the arm was involved to the elbow and the wall of the chest to the level of the twelfth rib. The lesion of the chest extended to involve all the left wall of the chest anteriorly. The sloughing continued over four and a half years, and during this time there was involvement of the entire anterior thoracic wall, the regions up over the right shoulder and the left shoulder and the left side of the back to within 2 inches (5 cm.) of the spine. The left arm was involved from shoulder to elbow. She also had osteomyelitis of the left humerus, which had to be drained and which cleared up rather readily. The aforesaid areas showed sloughing and healed by marked overgrowth of scar tissue. All forms of accepted treatment were tried, and none seemed to have any effect. The condition came "without any known cause and terminated after four years without apparent cause."

All laboratory tests failed to give a clue to the etiologic factor.

DULIN, Iowa City.

CONGENITAL ICHTHYOSIS (COLLODION SKIN). R. P. SEITZ, California & West. Med. **44**:500, 1936.

A report of a case of congenital ichthyosis appearing at birth is presented, together with a discussion of the literature. The infant is described in detail as to physical appearance and development. The fact that the skin continued to desquamate abundantly throughout the first year makes Seitz consider that retention of the epitrichium could not conceivably be the cause of the persistent and universal shedding. A biopsy of the skin at 9 months revealed hyperkeratosis and parakeratosis, evident in all layers.

MARSHALL, San Francisco.

THE PROBLEM OF THE ASTHMATIC CHILD. FREDERICK W. SCHLUTZ, Illinois M. J. **69**:33 (Jan.) 1936.

In a review of the accepted conception of juvenile asthma Schlutz states that from the standpoint of symptoms, three principal forms, or types, of asthma may be recognized in childhood: asthmatic bronchitis, characteristic of the period of infancy and observed frequently after the second year, true asthmatic attacks beyond that period and up to puberty, resembling in every symptom the adult type, and asthmatic cough, observed generally beyond infancy, characterized by a specific sound, extraordinary duration, resistance to medication and remarkable freedom of the lungs from any inflammatory change.

A striking feature of nearly all forms of asthma in childhood is their peculiar relationship to the manifestation of exudative diathesis and to lability of the nervous system. This is the constitutional phase which colors the background of practically every asthmatic person. All evidence indicates that asthma is practically always an allergic reaction.

The most important predisposing cause of asthma and of practically all allergic conditions is heredity. Food becomes relatively unimportant as a sensitivity factor after the fifth year. Then the air-borne substances are in the ascendant and allergens are derived from focal infections. In asthma due to infections, the causative factor is invariably located in the upper part of the respiratory tract.

Protein sensitization alone does not entirely explain all the manifestations associated with allergy. The theory has been advanced that the basis of allergy is a disturbed balance between the sympathetic and the parasympathetic nervous system. When the physicochemical balance is profoundly disturbed, various nonspecific and specific factors acting together will produce asthma and precipitate the attack.

The striking characteristics of the asthmatic state in childhood are persistent chronic cough and dyspnea during the actual attacks. Eosinophilia may be present

and marked or may not be present. The pathologic picture in the lungs is definitely noninflammatory.

The treatment of asthma is a complex problem and involves a number of factors. Schlutz expresses the belief that it is of prime importance to discover the specific allergen or allergens which may be etiologic factors in a given case and if possible to have them removed from the environment. If this is difficult or if such important foods as milk and eggs are involved, he believes that gradual desensitization can be brought about by the injection of specific allergens.

Asthma precipitated by allergens originating in focal infections are probably the most common type the practitioner will encounter. An early and thorough tonsillectomy and adenoidectomy will, he thinks, bring a great deal of relief. When infection of the sinuses is the focus, the situation is more difficult. Autogenous vaccine may be of some value.

For the acute attack Schlutz prefers epinephrine, ephedrine or perhaps atropine, and as a last resort change of climate. BARBOUR, Peoria, Ill.

STUDIES IN THE ECZEMATIZING PROPERTIES OF SOAPS. JAMES W. JORDON, HAROLD L. WALKER and EARL D. OSBORNE, New York State J. Med. **36**:791 (May 15) 1936.

Soap is definitely the cause of contact eczema in some cases and a contributing factor in many others. Patch tests are of great value in making the diagnosis. Studies of patch testing are reported. AIKMAN, Rochester, N. Y.

THE TREATMENT OF STAPHYLOCOCCAL SKIN LESIONS WITH TOXOID. L. E. H. WHITBY, Lancet **1**:1454 (June 27) 1936.

Whitby found that staphylococcic toxoid injected intramuscularly gave good clinical results in a high proportion of patients with recurrent and resistant furunculosis and was useful in the treatment of styes and carbuncles. The optimal dose of toxoid was found to be an individual matter. LANGMANN, New York.

CUTANEOUS TEMPERATURE IN THE VERTEBRAL REGION. JEAN SAIDMAN, Ann. Inst. d'actinol. **10**:92 (Feb.) 1936.

Study of the temperature in the vicinity of the vertebral column yields interesting information requiring intelligent evaluation. Spinal diseases cause changes not to be confounded with physiologic variations. Pott's disease tends to elevate the temperature over the median line of the spinal apophyses while it lowers that of the adjoining muscles. Nontuberculous spondylitis increases both the temperature at the median line and that of the lateral muscles. Diseases of the hip joint bring about changes affecting not only the lower limbs but the spinal column. There is evidently a heat-regulating mechanism which correlates the variations in temperature at the center, whatever the lesion.

VINTON, New York.

PROPHYLAXIS IS IN ALLERGIC DISEASES. VOGT, Bull. Assoc. internat. de pédiat. prevent. **3**:12, 1936.

As it is principally in persons predisposed thereto by heredity that allergic diseases manifest themselves, it seems necessary that before marriage both parties should be examined with a view to establishing whether or not such hereditary tendencies exist.

Nursing mothers must not make excessive use of such articles of diet as eggs and fish, which Vogt's observations convince him are likely to provoke allergic manifestations.

Precautions must also be taken not to include similar articles of food in the diet of the children of allergic families.

It should be noted that certain measures of general hygiene, for example, the provision of clean, dry and spacious dwellings, prevent, up to a certain point, the dangers consequent on excessive use of allergenic substances. Certain measures must also be taken in the domain of professional hygiene.

Stringent precautions must be exercised when it is found necessary to administer therapeutic injections of animal serums to persons who have already shown allergic tendencies or who belong to a family with a history of allergy.

AMESSE, Denver.

THE GEOGRAPHIC DISTRIBUTION OF ACRODYNIA. M. PÉHU, J. BOUCOMONT and LÉPANOFF, Rev. franç. de pédiat. **12**:589, 1936.

Complete information as to the geographic distribution of acrodynia is not obtainable, but from personal communications and the literature the authors have found that most of the known cases occurred in France, Switzerland, Holland, Great Britain, Belgium, Germany, Australia and the United States. The Balkan peninsula, Italy and Greece have had relatively few cases; also, the condition has been rare in the Scandinavian countries. The cases occur, as a rule, sporadically and in proportion to the population, being less frequent in large cities than in small towns and rural areas. From this study it seems certain that the condition does not occur in epidemic or pandemic forms.

LESLIE, Evanston, Ill.

A CASE OF DERMATOCHALASIS. MARCELLO GARCIA and AMADEU FIALHO, Arch. de pediat., Rio de Janeiro **7**:466 (Aug.) 1937.

A little girl who was suffering from this "loose skin" was small in stature. The authors had studied her for some time and so after death obtained a complete autopsy. The sections of the skin showed proliferation of the adventitia and elastic fibers. The thymus showed calcification of Hassall's corpuscles, and the thyroid, increase of colloid. The plates are well reproduced, and this article should be of interest to dermatologists.

SANFORD, Chicago.

THE USE OF DIATHERMY IN THE TREATMENT OF GENERALIZED PSORIASIS. V. A. LUKASHEV, Vrach. delo **18**:479, 1936.

Lukashev reports a typical case of severe generalized psoriasis of ten years' duration without remissions. Numerous methods of treatment had been used, but no improvement had been observed at any time. A course of generalized diathermy was then instituted. A complete clearing of the cutaneous process occurred after a course of twenty treatments, which were given every other day for periods of from twenty to forty-five minutes. A prophylactic course of treatments was given three months later. No recurrence of the disease had been observed up to the date of this report.

BODER, Los Angeles.

A CASE OF DERMATITIS EXFOLIATIVA RICHTER. NILS FAXÉN, Svenska läk.-tidning. **33**:763 (May 29) 1936.

A breast-fed infant was seen at the age of 10 days. Four days after birth his skin began to be inflamed, and when examined he had a typical exfoliative dermatitis. He was treated with sterile liquid petrolatum and was soon healed. One day before the boy's admission the mother had acquired an efflorescence below her right nipple, and this was found to be pemphigus. From this and from the infant's skin Staphylococcus aureus haemolyticus was cultivated.

WALLGREN, Göteborg, Sweden.

Surgery and Orthopedics

RUPTURED APPENDIX IN CHILDREN: ITS MANAGEMENT. C. J. BAUMGARTNER, California & West. Med. **45**:51, 1936.

One of the most serious problems in the management of acute appendicitis in childhood is the matter of making the diagnosis early. The failure of attending physicians to recognize the condition early accounted to a large extent for the fact that in 55 of a series of 100 consecutive cases in which patients with appendicitis were admitted to the Children's Hospital in Los Angeles the appendixes were found ruptured at operation. The causes of delay in diagnosis are summarized as follows: 1. Twenty-five of the ruptured appendixes were in children less than 5 years of age; in other words, the younger the child the less likely will its abdominal pain receive early serious consideration. 2. Not infrequently there was an associated respiratory disease which led to a mistaken diagnosis and for the treatment of which, in many cases, physics were administered. At least 29 of the 55 children presenting ruptures had been given physics. Baumgartner discusses briefly the surgical procedures and emphasizes, in the postoperative management, the value of the intravenous drip, the technic of which he outlines, and the value of the indwelling nasal catheter for the administration of fluids and food.

MARSHALL, San Francisco.

SIGNIFICANCE OF LOSS OF SERUM PROTEIN IN THERAPY OF SEVERE BURNS. D. O. WEINER, A. P. ROWLETTE and R. ELMAN, Proc. Soc. Exper. Biol. & Med. **34**:484 (May) 1936.

In forty patients with severe burns it was found that the serum protein values usually remained at from 6 to 7 per cent in spite of the marked concentration of the blood as noted by the erythrocyte count. The plasma proteins lost in the burned area contained more globulin relatively, as indicated by the ratio of albumin to globulin in the serum. Edema was often seen to develop in the unburned areas of the skin when intravenous fluids were supplied. In the severe cases, the kidneys excreted only from 10 to 20 per cent of the fluids administered. In a number of severely burned patients the red cell count decreased and the edema disappeared when intravenous acacia or blood plasma was given. These authors suggest that a diet high in protein with beef and soy bean meal may be helpful in these cases.

HANSEN, Minneapolis.

THE USE OF AVERTIN FOR THE PRODUCTION OF BASAL NARCOSIS IN CHILDREN. H. K. ASHWORTH, Arch. Dis. Childhood **11**:157 (June) 1936.

This study is based on a series of 5,918 administrations at the Booth Hall Hospital, Manchester, England. The advantages are: (1) The accepted dosage is safe; (2) the action is uniform and certain; (3) restlessness during the induction of unconsciousness is uncommon; (4) restlessness after operation is uncommon; (5) there is little appreciable effect on the chief organs of excretion.

The technic of administration is given in detail.

KELLY, Milwaukee.

ACUTE TOXEMIA OF BURNS: EXTRACT OF SUPRARENAL CORTEX IN TREATMENT. W. C. WILSON, G. D. ROWLEY and N. A. GRAY, Lancet **1**:1400 (June 20) 1936.

Acute toxemia, though only an occasional occurrence under present day treatment of burns with tannic acid, is still an important cause of death after extensive superficial burns, particularly those of young children. Regarding this toxemia no constantly available method of assessment is more accurate than observation of the signs of failing circulation. This phase begins at any time between six and fifty hours after injury.

The only characteristic pathologic change is severe necrosis and degeneration of the liver cells. The authors' experience of treatment includes not only various modifications of the tannic acid method but also other forms of local application; in

general measures, it embraces continuous as well as repeated intravenous infusion of physiologic solution of sodium chloride, gum in saline solution and dextrose in saline solution and also intravenous use of alkali. However, up to the time of the cases reported, if acute toxemia developed in a severe, fulminating form, it invariably proved fatal.

Three cases are reported in which an extract of adrenal cortex (eucortone) was used as an adjuvant in the treatment of established severe acute toxemia. In two of the patients a fatal issue within a short time was practically certain. The evidence of benefit from the use of the extract was clear. The mode of action of the extract in combating the toxemia is not clear. It seems to promote increased efficiency of the circulation. Pathologic studies have revealed little evidence of damage to the adrenal glands. The quantities of extract required for maintenance of circulatory efficiency are evidently considerable.

LANGMANN, New York.

CLINICAL DIAGNOSIS OF STRANGULATED DIAPHRAGMATIC HERNIA. L. GAROT, Arch. de méd. d. enf. **39**:337 (June) 1936.

Roentgenologic studies have shown that diaphragmatic hernia is much more common than formerly believed. Of 650 cases recorded in the entire literature, a review of which was published by Giffin in the *Annals of Surgery* in 1912, only 6 concerned children under 6 years of age, and only 15 were recognized during life.

From 1912 to 1928, however, Greenwald and Steiner collected 881 cases in subjects under 10 years, of which 27 were detected during life.

Garot stresses the importance of bearing these facts in mind when one is confronted with a patient showing unusual clinical phenomena in the left side of the thorax or in the abdomen. In many instances the hernia is tolerated so completely that its presence is unsuspected, and the abnormality is found only through roentgenograms made for some intercurrent disorder; in other cases the symptoms are limited to functional rather than specific manifestations. In cases of hernia involving the stomach, pain and vomiting are common, with strangulation always a possibility. Differential diagnosis is difficult; Garot could find but 6 observations in the past twenty years, and to this series he adds a further case, which is discussed in detail.

AMESSE, Denver.

SURGICAL QUESTIONS OF PEDIATRICS IN PRACTICE. ERWIN GOHRBANDT, Arch. f. Kinderh., supp. 8, 1936, p. 1.

Gohrbandt discusses the symptoms, diagnosis and indications for and contraindications to surgical intervention in surgical conditions of childhood under the following headings: harelip, cleft palate, spina bifida, spina bifida occulta, pleural empyema, pylorospasm, intestinal invagination, appendicitis, pneumococcic peritonitis, tuberculous peritonitis, inguinal hernia, femoral hernia and hernia of the abdominal wall, hydrocele, occurrence of hernia and hydrocele together, umbilical hernia, cryptorchidism, phimosis and paraphimosis.

CRANE, Chicago.

TREATMENT OF FLATFOOT BY EXERCISE. O. KOLB, München. med. Wchnschr. **83**:774 (May 8) 1936.

Strengthening of the muscles is essential in the treatment of flatfoot. Kolb calls attention to an exercise which was first described by Silberhorn and which necessitates use of a simple apparatus. The patient sits in a chair with his knees held apart by a wooden board. The feet are placed parallel at a distance of from 15 to 20 cm., and the toes are placed in holders to which springs are attached. These springs draw the toes outward, and the exercise consists of resisting this pull by drawing the toes inward. When the toes are drawn inward, the inner aspect of the foot is raised and the sole arched. At the beginning of the exercises pain occurs in the muscles which elevate the arch. It is possible to teach children to perform the exercise without supervision.

BRAHDY, Mount Vernon, N. Y.

Teeth and Dentistry

INFLUENCE OF DIET ON CARIES IN CHILDREN'S TEETH (FINAL REPORT). COMMITTEE FOR THE INVESTIGATION OF DENTAL DISEASE, Medical Research Council, Special Report Series no. 211, London, His Majesty's Stationery Office, 1936.

As stated in the preface of the report, the object of the investigation was, "to test the validity of the theory that a dominant factor in determining the structure of the teeth and their resistance to decay was the nutritional condition of the body, and more particularly the supply of specific food elements afforded to the teeth in early life." In all, 1,550 children were observed, but unfortunately, because of unavoidable conditions, the data on only approximately one fourth of this number could be included in the statistics reported. The subjects belonged to the lower economic levels and were observed in governmental institutions.

The study was comprised of three distinct investigations: The first concerned children in three separate institutions to whom treacle, olive oil and cod liver oil were given, respectively. These three groups numbered 44, 53 and 75 in the order given. The children of this portion ranged in age from 5 to 14 years, the average being 9½. They were observed at intervals of six months for a period of three years.

The second study concerned two groups of children in one institution having the same basic diet, the first receiving in addition olive oil, the second vitamin D. There were 59 and 60 children, respectively. The intervals of observation were identical, and the age levels were approximately the same as in the first study.

The third invesigation concerned children who at the termination of the period ranged between 2 and 5 years of age. They were divided into three groups: The first, in which there were 31, received treacle; the second, which included a like number, was given vitamin D, and the third, numbering 35, was given cod liver oil.

The results of all three investigations in the words of the committee are as follows: "The investigations described in this report show conclusively that a relatively high vitamin D content of the food can do much to diminish the incidence of caries if the vitamin is given during the development of the teeth; that a beneficial effect may be obtained if the vitamin is given at a fairly late stage of development; and that even when it is given after the eruption of the teeth, the onset and spread of caries is delayed."

The numerous tables which summarize the findings in the several studies indicate that there was relatively less caries in the children who received cod liver oil or vitamin D and there was relatively greater caries in those who received treacle. Relying on the work of Mrs. Mellanby, the committee chooses to interpret these findings as an indication that there was improved structure of the teeth in the children who received the vitamin and that this improved structure was responsible for the decrease in the incidence of caries. While there is no question but that these figures indicate a less incidence of caries in children receiving vitamin D, and this fact has been brought out by other investigations, it does not necessarily follow that this change in susceptibility is due to a change in tooth structure, provided this occurs.

The tables show with few exceptions that the group to the diet of which carbohydrate was added showed caries increased above that of the group fed the neutral substance. This appears to bear out the contention of those investigators who feel that the addition of carbohydrate (in this case, in the form of treacle) increases the susceptibility to caries. It has been the feeling of some investigators holding this view that the increased susceptibility is not related to the tooth structure but to the effect of the food substances on the environment of the tooth. This may be directly as it passes through the mouth or secondarily as the metabolic processes of the subject are affected. The possibility of the cod liver oil or vitamin D supplement acting in this manner must be considered irrespective of

the effect which it may have on the substance of the tooth. It has been repeatedly observed that teeth which present a finely calcified structure microscopically do decay, and it has never been demonstrated that the use of vitamin D or any other food substance would prevent dental caries in all persons.

The impression is distinctly given in this report that the decrease in the amount of dental caries found in newly erupted teeth, i. e., teeth appearing during the time of this investigation, was related to the fact that they were in the formative period during the time at which the vitamin was administered and consequently had a greater opportunity to improve their structure. The figures for the cod liver oil group as shown in the authors' tables XXXII and XLI do not indicate great divergence in the percentage of carious first permanent molars at first observation as compared with those erupted during the course of the investigation. It would be interesting in this connection to know the incidence of caries in the first permanent molars erupted within an interval of time similar to that of this study, also to compare the ages of these children.

A consideration pertinent to this investigation is the incidence of dental caries at different ages. Reliable studies of this indicate that the incidence is not as great at the time of the eruption of the first or of second molars as it is a year or two after. If this assumption is correct, one would not expect to find as great an incidence of dental caries in the newly erupted first and second permanent molars as in the corresponding teeth which were in the mouth at the time the investigation was begun.

It appears that this study confirms other investigations which indicate that the administration of cod liver oil decreases relatively the incidence of dental caries and that the administration of carbohydrates relatively increases dental caries, but it does not furnish irrefutable evidence that these changes are due to changes in the structure of the teeth, nor does it prove that the importance of administering the vitamin is directly proportional to the degree of development of the dental unit.

NOYES, Chicago.

THE EFFECT OF DIET UPON DENTAL CARIES IN THE SOUTH AFRICAN BANTU. P. ORANJE, J. N. NORISKIN and T. W. B. OSBORN, South African J. M. Sc. **1**:57 (Sept.) 1935.

This presentation is a report of three investigations designed to study the effect of particular European foods on the incidence of dental caries in relatively primitive South African natives between the ages of 16 and 30. In one study the Bantu natives (465) living on traditional native diets showed a definitely lower percentage of persons with caries (36 per cent) than did 90 members of the same tribe who had worked in the mines and been fed on European foods (56 per cent), and a still higher percentage (68 per cent) was found in town natives of various tribes. The average number of carious teeth per mouth as well as the average number of carious teeth per "carious mouth" contributed comparable proportionate figures.

Of 254 natives, those eating European foods for not more than six months were found to have less caries (39 per cent) than "farm boys" (43 per cent) and "town boys" (47 per cent). And in a third series of Bapedi natives, of the 50 women who remained in the native villages and lived on primitive diets, 22 per cent were found to have caries, while of 32 males from the same village who worked intermittently in European employ, 44 per cent had caries.

An attempt to correlate the influence of particular foods indicates that the caries increases with the consumption of sugar and to a lesser degree with machine-ground mealie meal. Those who received no vegetables had a somewhat greater incidence of caries, while in the persons recorded as taking sour milk regularly the condition was diminished. Oral hygiene did not appear to be of significance.

NOYES, Chicago.

Miscellaneous

THE RATE OF ABSORPTION OF SALICYLATES AND THE EFFECT OF CERTAIN COMPOUNDS ON THE RATE OF ABSORPTION OF ACETYLSALICYLIC ACID FROM THE STOMACH AND INTESTINE. W. B. BRADLEY, J. G. SCHNEDORF and A. C. IVY, Am. J. Digest. Dis. & Nutrition **3**:415 (Aug.) 1936.

The following conclusions were derived after a study on dogs both anesthetized and unanesthetized.

"1. Salicyl compounds are readily absorbed from the stomach.

"2. Gastric absorption is more rapid with the smaller salicyl molecules and at lower pH's.

"3. Calcium gluconate which does not materially affect the pH of the acetylsalicylic acid solution likewise does not affect the rate of gastric absorption.

"4. Sodium bicarbonate and magnesium oxide which raise the pH of the solution inhibit the rate of gastric absorption of acetylsalicylic acid.

"5. The rate of intestinal absorption of salicyl compounds is less affected by the pH of the solution than is their rate of gastric absorption.

"6. Substances used to protect against gastro-intestinal irritation, such as calcium gluconate, sodium bicarbonate, and magnesium oxide, do not markedly influence the rate of intestinal absorption of acetylsalicylic acid.

"7. Sodium salicylate, calcium salicylate and salicylic acid are absorbed from the intestine at a faster rate than is acetylsalicylic acid."

MORRISON, Savannah, Ga.

GENERAL MEDICINE AND PEDIATRICS. W. W. ANDERSON, Arch. Pediat. **53**:361 (June) 1936.

Anderson in an address before the Florida Pediatric Society mentions the many encroachments that have been made on pediatrics by various lay and public health organizations. Despite these intrusions there will always be a place for the trained pediatrician.

ORR, Buffalo.

AN OUTBREAK OF ARSENIC POISONING. Health News **13**:89 (June 8) 1936.

Eleven members of three Italian families are reported as having been poisoned with arsenic as a result of eating olives that had been packed in a barrel which had probably at one time been used as a container for spraying materials. The brine contained 0.5 per cent arsenic, and scrapings from the barrel contained 1 per cent. No deaths occurred.

AIKMAN, Rochester, N. Y.

REPORT OF SPIDER BITE. H. L. SHAW, J. South Carolina M. A. **32**:155 (June) 1936.

A Negro 20 years of age was bitten presumably by a black widow spider. He had typical symptoms and signs of spider bite. He was given calcium chloride intravenously, then morphine sulfate; there was little improvement, so magnesium sulfate was given intramuscularly. Prompt recovery followed.

WESTON JR., Columbia, S. C.

PEDIATRIC THERAPEUTICS. H. H. PERLMAN, Pennsylvania M. J. **39**:707 (June 15) 1936.

A study of the drugs officially listed in the United States Pharmacopeia discloses the fact that approximately 60 per cent of the medicinal preparations are of pediatric importance. Children show differences in recuperative powers, nervous system, development of the brain, cutaneous reactions, growth, height, emo-

tional temperament, and muscular and digestive systems. As a result of these variations, pediatric therapeutics is rendered more difficult. Special thought must be given to modification of unpleasantly tasting remedies, tolerance, idiosyncrasy and posologic variation according to age and weight. Pediatricians desire the benefit of pediatric counsel on the revision committees of both the United States Pharmacopeia and the National Formulary. Therapeutic instruction should be a part of the pediatric curriculum of medical schools. The tendency to undermine official remedies is discussed. Restriction of the sale of drugs and therapeutic remedies potentially dangerous to infants and children without a written order from a physician is desirous. The problem of posology is reviewed.

LOGUE, Williamsport, Pa.

NITRITE POISONING. S. R. NAIDU and P. V. RAO, Brit. M. J. **1**:1300 (June 27) 1936.

A boy 15 years of age died of a peculiar condition. It was found that he had in his possession a powder consisting of potassium nitrite. The signs, symptoms and biologic chemistry of poisoning with this substance are discussed.

ROYSTER, University, Va.

SOME COMMON DISEASES IN INFANTS AND CHILDREN. H. T. ASHBY, Brit. M. J. **1**:1312 (June 27) 1936.

A general discussion is given of some common diseases and conditions, particularly with reference to wasting, hemorrhagic diseases and intracranial injuries.

ROYSTER, University, Va.

THE RATIO OF LARGE TO SMALL LYMPHOCYTES IN PERSONS EXPOSED TO A LEAD HAZARD. D. O. SHIELS, M. J. Australia **1**:847 (June 20) 1936.

In a study of lead poisoning Shiels found that the ratio of large lymphocytes plus monocytes to the small lymphocytes more closely paralleled the clinical condition of the patient than the commonly used stipple cell count. In unexposed subjects he found the ratio to be 1.2:1. Following exposure there was an increase in the ratio to 3.3:1. So long as the ratio remained above 2:1 there was little danger that signs of lead poisoning would appear, but with clinical lead poisoning the ratio fell below 2:1 when the condition was less severe and below 1:1 when it was more severe.

GONCE, Madison, Wis.

SIMPLE MAMMARY HYPERTROPHY IN A GIRL EIGHT YEARS OLD. ROBERT DEBRÉ, JULIEN MARIE and JEAN BERNARD, Bull. Soc. de pédiat. de Paris **33**:460 (June) 1935.

In a girl 8 years old marked symmetrical hypertrophy of the breasts developed over a period of seven months. The breasts had the appearance of adult lactating breasts, but no secretion could be expressed from them. The child was apparently normal for her age otherwise.

BENJAMIN, Montreal, Canada.

THERAPEUTIC VALUE OF VARIOUS ARTIFICIAL SOLUTIONS IN INFANTS. E. LESNÉ, CLÉMENT LAUNAY and G. LOISEL, Bull. Soc. de pédiat. de Paris **33**:712 (Dec.) 1935.

Observations were made on ten dehydrated infants. Seven showed an increase in the chloride content of the corpuscules. The chloride content of the plasma was variable but usually about normal. The ratio of the chloride content of the corpuscules to that of the plasma was raised. In the severely dehydrated infants with acidosis the alkaline reserve was low, and the prognosis was bad. The

chloride content of the blood was determined at the beginning of treatment, and if it was low, from 5 to 10 cc. of a 20 per cent salt solution was injected intravenously. Subcutaneous injections of physiologic solution of sodium chloride, isotonic dextrose solution and isotonic solution of sodium bicarbonate were used. The latter, in doses of from 60 to 150 cc., was well tolerated and is considered a better fluid therapeutically than dextrose solution; the fear of alkalosis following its use is accorded little consideration. A transfusion of from 30 to 40 cc. of blood was given when the administration of an artificial solution alone was not followed by rapid improvement in the condition of the infant.

BENJAMIN, Montreal, Canada.

PHYSICAL AND MENTAL RETARDATION: PROBABLE RÔLE OF ATTEMPTS AT ABORTION. H. GRENET, PIERRE-PAUL LÉVY and P. ISAAC-GEORGES, Bull. Soc. de pédiat. de Paris **33**:730 (Dec.) 1935.

A boy 8 years old presented a somewhat senile appearance. He was dwarfed, microcephalic and markedly retarded mentally. It is thought that unsuccessful attempts at abortion on the part of the mother probably affected the child in utero adversely.

BENJAMIN, Montreal, Canada.

PHYSICAL THERAPY IN PEDIATRICS. LEONIDAS DETSI FILHO, Arch. de pediat., Rio de Janeiro **7**:443 (July) 1936.

Heliotherapy, electrotherapy, hydrotherapy, phototherapy and roentgenotherapy are discussed.

SANFORD, Chicago.

CANCER IN CHILDHOOD. EDUARD KLEMT, Monatschr. f. Kinderh. **66**:406 (Aug.) 1936.

This article contains brief reports of three cases of cancer—one case of cancer of the skin and two cases of cancer of the intestine. In the latter two instances the diagnosis was established only post mortem. Although the symptoms were reasonably clear, the diagnoses were overlooked because the pediatrician is not on the alert for such conditions. In the two cases of intestinal carcinoma the erythrocyte sedimentation did not show the increase that is found in adults.

GERSTLEY, Chicago.

RELATIONSHIP BETWEEN BODILY DEVELOPMENT, CONSTITUTIONAL FORM, TALENT AND CHARACTER AT SCHOOL AGE. EUGENE SCHLESINGER, Ztschr. f. Kinderpsychiat. **2**:161 (Feb.) 1936.

Five hundred and sixty boys in technical school (*Oberrealschule*) and seven hundred girls in high school (lyceum) at Frankfurt-on-Main, Germany, were studied with a view to correlating their bodily development, constitutional form, talent (educational) and character. The criteria for bodily development were nutrition, color of the skin and the mucous membranes, posture, etc. These characteristics were classified as from very good to good, from good to fair and from fair to poor. Constitutional types were grouped under the headings, (1) thin or asthenic, (2) medium, muscular or athletic and (3) broad, mesosomatic or pyknic. Talent was judged on the basis of achievement in school, including grades in German, foreign languages and mathematics.

The results are presented in tabular form according to the relation between the following elements: age and intelligence; general bodily condition and intelligence; habitus and intelligence; constitution, habitus, muscular power and intelligence; bodily strength and intelligence, and general bodily development and activity. These results are expressed in group numbers which do not lend themselves to abstracting.

JAHR, Omaha.

Society Transactions

AMERICAN PEDIATRIC SOCIETY

HENRY F. HELMHOLZ, M.D., *President*

Hot Springs, Va., April 29-May 1, 1937

HUGH MCCULLOCH, M.D., *Secretary*

First Session

Thursday Afternoon, April 29

INFECTION OF THE RENAL PARENCHYMA FROM THE PELVIS OF THE KIDNEY: PRESIDENT'S ADDRESS. DR. HENRY F. HELMHOLZ, Rochester, Minn. This paper appears in this issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN (**54**:1 [July] 1937).

ACIDOSIS AND ALKALOSIS IN CHILDREN. DR. GLENN E. CULLEN and DR. A. GRAEME MITCHELL, Cincinnati.

Experience with conditions of disturbed acid-base balance has led to the conviction that severe alkalosis exists much more often than has been previously recognized, especially in infants. It is so much more difficult to distinguish alkalosis from acidosis by physical examination in infants than in adults that we believe the diagnosis is not complete without data on the blood. Treatment for both alkalosis and acidosis must be on a calculated, quantitative basis, and its success is related inversely to the duration of the condition before treatment is started, especially in the case of alkalosis. In the treatment of the acidosis and dehydration of enteritis, it is important in the use of fluids to consider all fluids received by the child, else too much fluid may be used, resulting in edema.

In Cincinnati gastro-enteritis is much more prevalent than is ordinarily recognized. In 1936, of 4,350 patients admitted to the Children's Hospital, 220 suffered from a condition which had been diagnosed as gastro-enteritis (exclusive of 26 who had dysentery). The mortality was 16 per cent. In 23 patients (10 per cent) the initial carbon dioxide content of the blood was below 25 volumes per cent; of these, 7 died. In 34 it was between 25 and 45 volumes per cent; of these, 13 died, usually of some condition accompanying the gastro-enteritis. During 1936, 3 patients were admitted with severe alkalosis due to excessive administration of sodium bicarbonate; all 3 died. Among patients with conditions other than gastro-enteritis there were, during the same period, 7 with the carbon dioxide content below 25 volumes per cent, 4 of whom died, and 18 with between 25 and 40 volumes per cent, 10 of whom died.

EFFECT OF HYPERTHERMIA ON THE DISTRIBUTION OF WATER AND ELECTROLYTE IN THE BRAIN, LIVER AND MUSCLE. DR. HERMAN YANNET (by invitation), New Haven, Conn.

The investigation was planned in order to study two factors: (1) the distribution of water and electrolyte in tissues during the period of rapid growth and (2) the effect of hyperthermia on animals of different age groups.

The technic followed was that described by Harrison, Darrow and Yannet (*J. Biol. Chem.* **113**:515, 1936). Kittens were used as the experimental animals. The extracellular and the intracellular distribution of water and electrolyte were determined in a series of 20 normal kittens ranging in weight from 300 Gm. (about 4 weeks old) to about 2,500 Gm. (over 20 weeks old). Another series

of 20 animals, with the same range of weights, was studied similarly after their body temperature was raised 3 to 5 C. by exposure in a heating cabinet. The following results were obtained:

1. In the brain and muscle, growth is associated with progressive decrease in the proportion of extracellular fluid. In the brain, this decrease is probably secondary to deposition of myelin. In the muscle, this decrease is related to enlargement of the muscle cells. In the liver, growth is associated with equal increase in all the elements of the tissue so that their proportions remain constant.

2. As a result of hyperthermia, significant changes could be demonstrated only in the brain. Here there was redistribution of water, resulting in shrinking of the cells and increase in the amount of extracellular fluid. Associated with the diminution of intracellular water there was concentration of the intracellular elements, namely, potassium, nitrogen and phosphorus. Since the term "edema" ordinarily implies accumulation of extracellular fluid, one may, perhaps, speak of this change as edema of the brain. However, it is a peculiar type of edema in that there is no increase in the total amount of water in the brain.

DISCUSSION

DR. A. A. WEECH, New York: A new point of view brought to bear on an old problem is always helpful; in this presentation there is a new concept of the cause of the convulsions which occur with fever in children. It is even nicer that the concept arises from the application of a new method and that the method gives promise of aid in the elucidation of many other problems.

There is just one point which troubles me in these calculations of the volumes of extracellular and intracellular fluid. The calculations depend on the assumption that all of the chloride in a tissue is outside the cells. Certainly, so far as the whole body is concerned, the chloride must be largely extracellular. In muscle the chloride appears to be almost entirely extracellular. However, it is known that chloride exists within the red blood cell. If this is true, may it not be that other organs—for instance, the liver or the brain—are also exceptions to the general rule and contain intracellular as well as extracellular halogen? The possibility that such exceptions may occur makes one wish for confirmatory evidence of the distribution of fluid between cells and surrounding spaces when the distribution is calculated in this way. I was glad, therefore, to see the photomicrographs of the brain, which appear to support the suggestion of chemical analysis that hyperthermia leads to loss of water from the brain cells.

DR. HERMAN YANNET, New Haven, Conn.: At present there is no direct way of proving the assumption that all the chloride in the body except that in the red blood cells is extracellular. For proof one must depend on the reasonableness of the results obtained by the application of the assumption. Because these results seem to agree with the accepted knowledge of acid-base equilibrium as well as with the more recently determined facts as to the distribution of water of the body one may assume that for all practical purposes there can be little, if any, intracellular chloride.

For instance, in the muscles of adult animals, about 10 per cent of the weight represents the quantity of water required to dissolve all the chloride in the tissue at the concentration assumed to be present in the extracellular fluid. Of this amount, about 5 per cent probably represents the plasma present in the tissue. The amount remaining, namely, 5 per cent of the weight of the tissue, is so small that to assume that even a small proportion of the chloride is intracellular would lead to an impossible result. In the other tissues, such as brain and liver, one depends more on the concentration of the intracellular potassium, calculated on the basis of the original assumption, to prove the reasonableness. To assume that even a small proportion of the chloride is intracellular would yield values for intracellular potassium which are highly improbable. Thus, while one cannot analyze intracellular water to prove the exclusively extracellular position of chloride, to assume any other arrangement would lead to results most contradictory to the accepted knowledge of acid-base equilibrium and to the distribution of the water of the body.

FACTORS INFLUENCING THE DISPOSITION OF FLUIDS GIVEN INTRAVENOUSLY. ANN S. MINOT, PH.D. (by invitation), Nashville, Tenn.

Some infants with dehydration and intoxication resulting from severe diarrhea present abnormalities in the concentration of protein in the serum, in the permeability of the capillaries and in the equilibrium between fluid and electrolyte. The inability of these infants to dispose normally of fluids introduced intravenously makes it difficult to restore them to a normal state of hydration. One often sees, as venoclysis continues, a concentration of the cellular elements of the blood even in the absence of transfusions of whole blood, and at the same time, a progressive reduction in the concentration of protein in the serum. There is gradual loss of plasma fluid and protein through the capillaries, in which the permeability to the colloids of the plasma is increased. Gross edema results, the urinary output remains scanty, and the circulation becomes progressively impaired.

Studies are presented in which dogs with experimentally damaged capillaries were used as subjects. Such animals were given fluids at various rates by continuous venoclysis, both with and without transfusions of plasma.

The results obtained explain and emphasize anew the importance of the point made by other investigators that when fluids are given by venoclysis to markedly prostrated infants only conservative amounts should be given, and transfusions of blood or plasma should be given early, repeatedly and in liberal amounts. Additional means for controlling capillary leakage are being investigated.

DISCUSSION

DR. KATHARINE DODD, Nashville, Tenn.: I have followed Dr. Minot's experiments and the children treated by continuous venoclysis, whose course suggested some of the experiments. I wish to emphasize that the work is not used to discourage the use of intravenous drips. It has, however, proved very effective in two ways: First, one feels that one now has a much better idea as to why in dehydrated children "the more fluid the better" is not the best policy, especially when the fluid is given intravenously. Second, since the house staff has seen some of this work, it is easier to persuade them that a great deal of fluid sometimes does more harm than good.

Also, I wish to say that there is no proof that the children have any injury of the capillaries except that a certain number of them do exactly as the dogs do. Since seeing this work my associates and I have found that it is apparently quite useful to follow the hemoglobin in the children, and if there is concentration of the hemoglobin without transfusion of blood we know that we had better give serum intravenously and go slow in regard to intravenous introduction of fluids.

DR. HORTON CASPARIS, Nashville, Tenn.: I think that Dr. Minot's discussion probably explains to some extent what used to happen to seriously sick and dehydrated infants to whom doctors gave plenty of fluids and in whom they reestablished the disturbed acid-base equilibrium but who in spite of this died because the kidneys did not function. From her discussion it appears that the fluids leaked into or were drawn into the tissues rather than being poured out through the kidneys, and that is the chief explanation of why the kidneys failed to function.

Another point of interest in her paper is that in her experimental work certain drugs which had been recognized as disturbing the sympathetic-parasympathetic balance seemed to influence the passage of fluids through the capillaries. This might be of fundamental importance in explaining the possible relationship between what occurs in these children and changes in the sympathetic-parasympathetic balance. Drugs which influence the autonomic system may prove to be of practical value, but their clinical use cannot be advocated until a great deal more experimental work has been done.

DR. JOSEPH STOKES JR., Philadelphia: I hesitate to mention again serum desiccated from the frozen state, but in this particular condition I think it may be of considerable value. My co-workers and I have used such serum, redissolved in concentrated form and then filtered through a Berkefeld filter, in nephrosis, in particular, and also to decrease intracranial pressure. Hughes and his co-workers

have experimented with experimental traumatic shock in animals. Following marked loss of plasma protein through the capillaries they found that rapid recovery occurred with injections of concentrated plasma protein. In the condition mentioned by Dr. Minot serum desiccated from the frozen state should be of considerable value.

DR. BENJAMIN KRAMER, Brooklyn: Some time ago my colleagues and I attempted to increase the serum protein of children by repeated transfusions. We did not succeed in materially increasing it but produced a tremendous increase in the hemoglobin and in the cell volume, far above the calculated amounts.

In seeking an explanation for this phenomenon we came across reports of some experiments on animals in which it was shown that when blood is repeatedly transfused in large amounts during a brief period much of the plasma passes through the capillaries into the tissues. There was no increase of albumin in the urine to account for this loss of serum proteins. Apparently, plasma proteins can pass through the capillaries of the normal animal. Such a migration is therefore not necessarily an indication that the capillaries have been damaged.

DR. RUSTIN MCINTOSH, New York: Any one who has made determinations of blood volume is conscious of the sources of error inherent in the method. I should like to point out a feature of this investigation about which I think Dr. Minot has been perhaps a little too modest.

If the dye method is used and if there is loss of dye through the capillaries because of the injury deliberately induced in her experiment, the error is likely to be in the direction of a false increase in the calculated volume of the plasma or of the blood.

Therefore, her results show indubitably that damage of the capillaries causes loss of plasma protein, because if the permeability of the capillaries produced an error by allowing the dye to escape, this error would be in the reverse direction and would tend to neutralize the final effect. So she has been on the conservative side, and if her determinations are wrong, they are wrong in showing too small a loss of plasma protein.

I should like to ask whether she has any clinical or other criterion for determining when she should give a blood or a plasma transfusion in therapy. Is it a matter of the level of hemoglobin?

ANN S. MINOT, PH.D., Nashville, Tenn.: I think it is true that some plasma protein may be lost without damage of the capillaries necessarily having occurred. However, although I have not been able to present all the data, my associates and I made many control experiments in all this work, and the intoxicated animals, whatever the factor involved, lost a great deal more plasma protein much more rapidly than the control animals.

As for our criterion of when to give plasma, I do not think we have one. If during the administration of fluid we see the hemoglobin, at whatever level it may be, concentrating rather consistently in the absence of transfusions of whole blood we feel that at least plasma fluid and probably plasma protein are being lost from the blood stream. We feel then that administration of plasma is the most effective way of restoring protein. For this purpose plasma is far more effective than whole blood. Except an occasional child in whom anemia is a prominent part of the picture, the patient probably needs plasma protein much more acutely than he needs hemoglobin.

IRON-COBALT TREATMENT OF PHYSIOLOGIC AND NUTRITIONAL ANEMIA IN INFANTS.

DR. KATSUJI KATO, Chicago.

The evidence pointing to the possible catalytic action of cobalt in the metabolism of iron for the synthesis of hemoglobin and the production of erythrocytes in young organisms is based on the following observations:

1. The effective use of iron and cobalt in the treatment of physiologic and nutritional anemia in infants.

2. The rapid and favorable regeneration of hemoglobin and multiplication of erythrocytes in anemic young animals under iron-cobalt feeding.

3. The histology and morphology of the blood and blood-forming organs in infants and experimental animals fed iron and cobalt.

4. The results of chemical analysis of certain organs (liver, heart and kidneys) removed from anemic animals treated with iron-cobalt medication.

The presentation will be accompanied by appropriate graphs, tables and charts in lantern slides. A series of camera lucida drawings illustrating the cell morphology and the histology of hematopoietic organs, as well as photomicrographs, will be exhibited.

Incidentally, a newly devised combination hemopipet which has been found to be of great usefulness in the present research on the blood of infants and small laboratory animals will be presented and demonstrated.

DISCUSSION

DR. ALFRED H. WASHBURN, Denver: I am much interested in this paper which Dr. Kato has just presented. He referred to the work of Orton, now at Yale University, but formerly at the University of Colorado. I had the privilege of observing some of Orton's original work with anemias of rats, in which he demonstrated the development of polycythemia following the administration of cobalt.

I should like to ask Dr. Kato what preparation of iron he used and whether he is sure it was free from copper. I understood him to say that Orton had given cobalt only in conjunction with both copper and iron. I believe I am correct in saying that in some of his work Orton used cobalt in combination with copper-free iron. It is well known that the usual preparations of iron contain at least traces of copper. I should like to ask Dr. Kato whether he can be sure that the effect which he has demonstrated is not dependent on iron plus copper plus cobalt.

DR. KATSUJI KATO, Chicago: In answer to Dr. Washburn's question I wish to say that the iron was given in the form of ferric ammonium citrate, which is said to be ordinarily contaminated with a trace of copper and a few other heavy elements. While I did not analyze the preparation quantitatively, it is assumed that copper, if present as a contamination, must be in an exceedingly small amount, whereas the amount of copper used by Orton in his experiments was far in excess of any quantity due to contamination.

AN UNUSUAL CASE OF HEMOLYTIC ANEMIA. DR. HUGH W. JOSEPHS, Baltimore.

A 9 year old boy had, since about 4 years of age, uveitis of obscure etiology, which eventually led to complete blindness. At 8 years of age, anemia was noted but no jaundice. Somewhat later he was given a course of treatment with neoarsphenamine, following which jaundice developed, being severe enough to necessitate his admission to the hospital. He was then found to be suffering from hemolytic anemia, which conformed in all details to congenital hemolytic jaundice. His family was examined, and one sister was found to have slightly increased fragility of the blood.

Five months later, on admission for splenectomy, his blood had undergone a profound change. There was marked erythroblastosis, and the cells instead of being predominantly microcytes were largely macrocytes. A tendency to hemolyze transfused blood was shown, and it was found impossible to raise the number of red cells above 1,500,000 per cubic millimeter. The erythroblastosis became gradually more severe, with one period of comparative remission during the use of whole liver extract. Eventually the spleen was removed, when it was evident that the child could not otherwise get well. For about three or four weeks the condition was much better than before the splenectomy; then the erythroblastosis recurred in an extreme form, and the child died.

From the point of view of pathogenesis the case was considered to be due to two processes: (1) a constitutional process similar to, if not identical with, that responsible for congenital hemolytic jaundice and (2) a second process leading to a profound and rapidly progressive disturbance in erythropoiesis.

Interest attached especially to the second process. Was it congenital and therefore essentially a manifestation of congenital hemolytic jaundice, though a rare one, or was it due to some acquired condition and therefore superimposed on the background of congenital hemolytic jaundice? It was felt that it would be very unusual for a constitutional anomaly to manifest itself in a process which came on as late as this and progressed as rapidly. It seemed more likely that the disturbance in erythropoiesis was due to some factor such as infection, intoxication or other acquired disease. The uveitis suggested infection as the most likely possibility.

The onset of the jaundice after the administration of neoarsphenamine introduced the possibility of intoxication, especially one involving the liver, but autopsy did not confirm this or point to any other recognized disease process which might account for the erythropoietic disturbance. Syphilis and tuberculosis were excluded. No conclusion could be reached from this study regarding the essential nature of the case, but similar cases, both in my own experience and in that of others, point to the existence of a problem for future investigation.

DISCUSSION

DR. THOMAS B. COOLEY, Detroit: My colleagues and I have been observing a case like Dr. Josephs' except that we have been unable to find any evidence of a hereditary or a familial element. Our patient was a girl of 6 years who came to the hospital a little more than a year ago with a story of four or five months' illness, the first indication of which seems to have been jaundice, which lasted about a month, then disappeared. This was followed by progressive weakness and anemia. On admission her blood showed about 4 Gm. of hemoglobin per hundred cubic centimeters, the microcytosis and increased fragility of hemolytic icterus, and about 10,000 normoblasts per cubic millimeter.

We watched her for three weeks, during which we gave her 1,000 cc. of blood by transfusion, which brought the hemoglobin up to 8 Gm. Splenectomy was then performed, following which she did rather better for a month. The hemoglobin rose rapidly at first, then fell to 8 or 9 Gm., and she began to go down hill. An abscess developed, apparently from a gland, which drained through the rectum, and the child was quite ill. That, however, did not last long. In the meantime, the normoblast count rose steadily, reaching 40,000 to 50,000 and remained at that level for some time.

The abscess finally healed in July. In August hemolysis was still going on, the hemoglobin was falling, and the normoblasts were increasing in number. As an accessory spleen had been removed at the first operation, the abdomen was reopened to make sure that another one had not been overlooked, which might be making trouble. The only finding was a general enlargement of lymph nodes throughout the abdomen, not hemolymph nodes, but nodes showing on biopsy only simple inflammatory changes. There was no accessory spleen.

The condition progressed in much the same way until December; at that time the hemoglobin had fallen to 5 Gm., and the child seemed gravely ill. She had been given iron by mouth, liver extract intramuscularly and occasional transfusions. In December, for no apparent reason, she began to improve, and by April of this year she had apparently made a complete recovery, in the course of which she has grown remarkably. At the beginning of hospitalization she was a puny child of 6. She now looks more like a well developed girl of 9 or 10. The blood is normal except that the microcytosis and fragility are unchanged. A difference between this case and that described by Dr. Josephs was that the normoblasts seemed to come in showers, sometimes disappearing from the circulation for some days.

DR. HUGH W. JOSEPHS, Baltimore: I asked Dr. Diamond to give me a short abstract of his cases. He said they were going to be discussed but sent enough to make me realize that his cases differed from mine in that they did not show evidences of congenital hemolytic jaundice. He said he did not think his cases belonged to that group; mine apparently did as far as we could make out.

The fact that these cases are not similar certainly is against there being any special entity to which a new name can be applied. I think it is necessary in

each case to determine just what the etiologic features may be, whether congenital or acquired, and especially what part infection plays in the development.

It may be, of course, that infection plays a special part in patients who are already subject to hemolytic anemia on a constitutional basis. A case was reported in the literature from France in which there was marked erythroblastosis along with hemolytic anemia. In that case, the child was very largely benefited by splenectomy but not followed for any great length of time before the report was made.

With regard to the disappearance of erythroblastosis, it might be remembered that on the second chart I showed there was almost complete disappearance of erythroblastosis during the treatment with liver. Whether that was a result of the administration of liver or just a spontaneous remission, I have no means of telling.

THE VITAMIN D REQUIREMENT OF THE CHILD. DR. P. C. JEANS and GENEVIEVE STEARNS, PH.D. (by invitation), Iowa City.

It has long been known in regard to animals that the weight of the water in the skeleton equals 20 per cent of the weight of the body throughout life. It has been shown also that the water content of the skeleton is in inverse ratio to the ash content, the water content being high at birth and low at maturity. The data on human subjects correspond to these findings very closely, and it is widely assumed that the same relationships occur. The water content of the human skeleton is known for various age periods. From all of these data the required retention of calcium for each period of growth may be calculated. It seems desirable to attain this requirement in the nutritional management of children. This minimum is used as the basis of comparison in the present report.

In the first year of life, if vitamin D is given, the retention of calcium increases with increase in the calcium intake, but without vitamin D little or no increase in the retention of calcium is observed, and in the majority of cases the retention is below the calculated minimum requirement at all levels of intake. When milk with 135 units of vitamin D to the quart is fed at the level of 1.8 ounces (53 cc.) to the pound (0.45 Kg.) of body weight, most infants show more than the calculated minimum retention, but with the smaller amounts of milk commonly fed many infants show retention below this minimum. The retention with 400 unit milk is greater than that with 135 unit milk and is above the calculated minimum for most babies at less than 1.8 ounces of milk to the pound.

In children over 1 year of age the retention of calcium increases with the intake whether or not vitamin D is given, but the retention is greater with vitamin D than without.

Between 1 and 6 years of age without vitamin D the retention in half of the cases is below the minimum requirement with 750 cc. of milk, and in the majority of cases it is not above the minimum until 1,000 cc. of milk is given. With 340 units of vitamin D daily the retention in the majority of cases is above the minimum requirement when the amount of milk taken is 750 cc., and that in all cases, when the amount taken is 1,000 cc. With 500 cc. of milk the retention in half the cases is below the minimum. Thus, between 1 and 6 years of age with the customary intake of milk vitamin D is necessary for many children, just as it is for many babies. It seems apparent also that 500 cc. of milk is not adequate for half the children even when a teaspoonful of cod liver oil is taken daily.

The child over 6 years of age requires vitamin D to a slightly less extent than the child under 6, though in many children without vitamin D the retention is below the minimum requirement when the milk taken amounts to 750 cc. or less. With 340 units of vitamin D daily, from 750 to 1,000 cc. of milk is adequate for most subjects; 500 cc. is insufficient for most children even with a teaspoonful of cod liver oil daily.

It appears that vitamin D is as important for the child as for the infant and that the total quantity required may be as great.

DISCUSSION

DR. HORTON CASPARIS, Nashville, Tenn.: I should like to know whether Dr. Jeans would advise giving more than 1 teaspoonful of cod liver oil a day.

DR. HENRY DIETRICH, Los Angeles: I should like to ask what time of the year these experiments were carried on, and is this fresh milk or a canned product?

DR. EDWARDS A. PARK, Baltimore: I should like to make this observation, which must be apparent to every one: Vitamin D obviously is not so essential to older children as to infants, because the older child is conspicuously free from rickets and the infant conspicuously liable to rickets. Any conclusion made without consideration of this fact has something serious the matter with it.

DR. P. C. JEANS, Iowa City: In reply to the first question: I have not studied retention in children receiving more than 1 teaspoonful of cod liver oil. However, I have found that in infancy approximately the maximum retention was obtained with a teaspoonful of cod liver oil daily. When more was given, the increase in retention was very little, not at all proportionate to the increase in vitamin D. I do not know if this would be true also for the child.

These experiments were conducted throughout the year. Even in the winter these children ran about and were out of doors as much as the average child living at home. The experimental conditions approximated the average conditions which obtain in the home. Fresh milk was used for all children beyond the first year.

Obviously, rickets cannot be used as a criterion for the vitamin D requirement of the older child. It has been my contention that rickets is not a suitable criterion even in infancy. Some criterion is necessary. One which seems to me to be satisfactory was presented. The failure of the older child to acquire rickets does not seem to be an indication that he may not be suffering from the detrimental effects of a deficiency in vitamin D, such as dental caries or deficient mineralization of bone.

Second Session

Thursday Evening, April 29

CRYPTORCHIDISM TREATED WITH GONADOTROPIC HORMONES. DR. JOHN A. BIGLER, (by invitation), Highland Park, Ill.; DR. L. MARTIN HARDY (by invitation), Chicago, and DR. H. V. SCOTT (by invitation), Fort Wayne, Ind.

This study was made on 96 boys ranging in age from 1 to 13 years; there were 89 with 113 undescended testicles, 5 with postoperative atrophy of the testicle and 2 with hypoplasia of the genitalia but without cryptorchidism.

We were able to compare the effects of two different gonadotropic principles on cryptorchidism and the genitalia: (1) that in the gonadotropic extract from the urine of pregnant women, which assayed 100 rat units per cubic centimeter, and (2) that in the gonadotropic extract from the anterior lobe of the pituitary gland, which assayed from 10 to 15 rat units per cubic centimeter. The injections were given intramuscularly or subcutaneously. A dosage of from 1 to 3 cc. was maintained in 42 patients, of from 3 to 4 cc. in 19, of from 4 to 5 cc. in 5 and of from 5 to 7 cc. in 5. Rest periods were given to 32 patients. Local or general reactions, varying from local erythema to generalized allergic manifestations, occurred in 32 boys. Some changes in behavior were occasionally noticed.

In 14 boys one or more of the changes which occur at puberty developed; 6 boys (aged from 12 to 13 years) showed growth of pubic hair and changes of voice; 4 (aged from 3 to 12 years), growth of pubic hair and enlargement of the penis, and 4 (aged from 4 to 11 years), only growth of pubic hair. In 5 patients hernia developed during treatment. There were never any changes in the urine, and the height and weight were not unduly influenced.

There was an increase in the size of normal-sized testicles in 17 patients, 9 of whom received the gonadotropic extract from the urine of pregnant women, the gland extract and 5 both extracts. Of 7 patients with small testicles, 3 showed an increase of the testicles to normal size. In 6 boys with postoperative atrophy

of the testicle no change occurred. Enlargement of a previously small-sized penis occurred in 5 of 15 boys, all 6 years of age or younger. In 8 boys, all 6 years of age or younger, there was an increase in size of a previously normal-sized penis. The penis has returned to normal size in all but 6 patients.

Treatment with the gonadotropic principle of the anterior lobe of the pituitary gland was given to 32 boys with 40 undescended testicles. Of these testicles 26 remained unchanged in location; 4 showed partial descent, and 10, or 25 per cent, complete descent. After treatment was discontinued, on further observation only 7, or 18 per cent, of the testicles remained descended. If those partially descended are included, 14, or 35 per cent, were benefited.

The gonadotropic extract from the urine of pregnant women was used in the treatment of 23 patients with 31 undescended testicles. No change was observed in 12 testicles; 4 partially descended, and 15, or 48 per cent, completely descended. On further observation only 14, or 45 per cent, remained descended. If those showing partial descent are included, 19, or 61 per cent, were benefited.

Both extracts were used in the treatment of 16 boys with 20 undescended testicles. In 9 of these testicles there was no change; 2 partially descended, and 9, or 45 per cent, showed complete descent. On further observation only 5, or 25 per cent, remained descended. If the partially descended testicles are included, 11, or 55 per cent, were benefited. By dividing the cases of undescended testicle into age groups it is found that descent occurred in 18 per cent of the 1 to 4 year group, 36 per cent of the 4 to 9 year group and 43 per cent of the 9 to 13 year group.

An untreated control group of 18 patients with 22 undescended testicles was observed over a similar period of time. In none did descent of the testicles occur.

DISCUSSION

Dr. Henry L. K. Shaw, Albany, N. Y.: Dr. Bigler and his associates have done an excellent bit of work, having had an opportunity to study so many cases, but I think one ought to differentiate between true cryptorchidism and what might be called physiologic retention of pseudocryptorchidism.

Dr. McLellan, a surgeon in London, published an article in the London *Lancet* to which he gave the title "Decline and Fall of the Undescended Testis." Twelve patients were referred to him by physicians experienced in children's diseases, and in every one of these patients he was able to reduce the offending testicle without operation.

My colleagues and I have been rather interested in this subject. Our endocrinologist, Dr. Hamilton, was able last year to secure some of the crystallized androgen from Switzerland. He started on monkeys. It was news to me that in monkeys the testicle does not descend until the age of 4 or 5 years. Starting with monkeys from 1 to 2 years old, with the crystallized androgen he brought about complete descent of the testicles inside of three or four weeks. He has done this in a dozen or more monkeys. We started to find some children. My associates went through the hospital files, choosing children from 1 to 13 years of age. They found 16 children with undescended testicles. When Dr. Hamilton looked at them more carefully, by applying hot packs for half an hour and gentle traction he was able to rule out 10 of the cases. So there are only 6 patients on whom he has been working. The results have been interesting.

Dr. Hamilton has been studying various gonadotropic substances, with the conclusion that they are not always of the potency described on the ampules. They are but extracts and for their effectiveness depend on a responsive testis to produce the androgen which he believes from his work in animals and man to be essential for testicular descent. Stable synthetic androgenic substance is available, in contrast to unstable gonadotropic extracts. A comparative study is being made of the usefulness of this synthetic chemical in both false and true cryptorchidism, and the results should be compared with the results obtained when the production of androgen is stimulated by gonadotropic extracts, as has been reported in this paper by Dr. Bigler and his associates.

Dr. Julius H. Hess, Chicago: An important question has been raised, I dare say, in the minds of all. Dr. Bigler has stated that in several of the cases there was at least temporary hypoplasia of the genital organs. My associates

and I have seen 39 cases and have not observed that condition in any of our cases. Our patients averaged older than those Dr. Bigler has spoken of. We have very few under 10 years of age. Recently at one of the hospitals in Chicago a patient was shown who had a very large penis. The observation was passed around among the genito-urinary specialists of the city as a horrible example of what the pediatricians had done to this child. At the same meeting there were reported 2 cases in which marked atrophy of a testicle or of the testicles had been found at operation.

In our series of 39 cases there were 28 in which the cryptorchidism was completely reduced. In 25 of these the condition was bilateral and in 3 unilateral. In 4 cases we described the result as partial reduction; in 1 of these the condition was bilateral, and in 3 it was unilateral. There were 7 failures; in 3 the condition was bilateral and in 4 unilateral. Of the failures in bilateral conditions, two concerned atrophy of the testicles, which, we believe, was due primarily to pituitary abnormality. Two of the four failures in unilateral conditions also had to do with marked atrophy of the testicle.

As has been said, one must distinguish those who show a real pathologic condition of the testicle before treatment is started, and usually one must depend largely on the type of child one sees before one in deciding whether the condition is due to an endocrine disturbance. The children who have bilateral cryptorchidism and are of the endocrine type offer the best chance of solution of the problem of treatment. Those in whom the cryptorchidism is unilateral, in whom the testicle cannot be found, are the very poorest for treatment.

We feel that a problem is raised if we don't see any benefit after administering from 2,500 to 3,000 units of the gonadotropic extract of the urine of pregnant women, and that is the dose we have favored. We have tried placental extract, extract of the anterior lobe of the pituitary gland and some of the gonadotropic extracts, but our best results have been with the extract from urine. When we do not see any result after giving from 2,500 to 3,000 units, we have discontinued the treatment. We also feel that in most instances one should hesitate to use it in the younger age group. It is difficult to say how many of those children when older would have had a spontaneous descent. Our friends who specialize in genito-urinary conditions claim that if we had left many of these treated testicles alone the testicles would have descended of their own accord.

The matter I should like to see discussed here is whether other members have had experience with hypoplasia of the genital organ, and if they have whether they got the recession Dr. Bigler spoke of after stopping the treatment. That is the thing the public wants to know more about and the thing the pediatrician should be informed about.

Dr. John A. Bigler, Highland Park, Ill.: As to Dr. Hess' question relative to enlargmeent of the penis as illustrated on the slides: In every child in whom the penis was smaller than normal before treatment and in whom enlargement greater than normal occurred there was a return to normal size after treatment was stopped. The children in this group were 6 years of age or younger.

In this group in which the penis was of normal size to begin with and enlarged during treatment there were only 2 who showed a return to normal size. In the other 6 the penis is still very much larger than normal; they were in the group of 6 years of age and under. We have had no experience of this type in the older children.

We operated on 20 of the children. In 2 the testicle was smaller than normal; in 4 it was atrophic and pea sized, and when it was brought into the scrotum no development took place. In 13 of the children small hernias were found, and in 8, definite adhesions that held the testicle in the canal.

No one has mentioned the fact that the function of the testicle when in the canal is not the same as when it is in the scrotum. At the Mayo Clinic a higher percentage of malignant changes was found in the undescended than in the descended testicles. In adults, undescended testicle occurs in about 2 of every thousand. There are a number of cases in which the testicle does not descend

into the scrotum during early childhood, although undoubtedly in many it descends into the scrotum spontaneously before puberty.

RATES OF GROWTH, OSSEOUS DEVELOPMENT AND MENTAL DEVELOPMENT IN CRETINS AND IN DWARFS. DR. LAWSON WILKINS, Baltimore.

The rates of growth, the osseous development and the mental development of dwarfs and cretins have been followed by means of special graphs in which height has been expressed as "height age" in order to make a comparison with "bone age" and "mental age." In young cretins before treatment there are (1) marked retardation of the bone age, (2) retardation of the height age to a less degree than the bone age and (3) mental retardation corresponding roughly to the retardation in height. Under thyroid therapy there are (1) rapid osseous development, which is the most striking effect, (2) less rapid rise in the height age than in the bone age and (3) rapid loss of torpor and initial stimulation of activity, followed by more gradual mental development. In some cases there is a permanent mental defect. Poor results are frequently due to delay in beginning therapy or to inadequate dosage of thyroid, but in some instances irreparable neurologic damage may have resulted at a very early age. The rapid disappearance of all the physical signs of cretinism on treatment makes it difficult to judge clinically the dose of thyroid necessary to fulfil the body's needs. The developmental charts are a guide to treatment. In order to stimulate a cretin sufficiently for him to attain a normal level of development, it is probably necessary to produce mild hyperthyroidism.

The developmental patterns of dwarfs have been studied by the same methods. A tentative classification has been made as follows:

Group I: marked osseous retardation; thick, stocky body build; dry skin and hair.

Group II: marked osseous retardation; symmetrical body build; normal skin and hair.

Group III: moderate osseous retardation (which is less marked than the growth retardation); symmetrical body build; normal skin and hair.

Group IV: normal development of bone; symmetrical body build; normal skin and hair.

There is evidence that the dwarfs of the first group suffer from hypothyroidism. It is pointed out that the knowledge concerning these groups is incomplete and that positive criteria of thyroid hypofunction are lacking. One is not justified at present in accepting osseous retardation as prima facie evidence of hypothyroidism. It is necessary to make complete studies of the basal metabolic rate, cholesterol, creatine metabolism and blood iodine curves, assays of the thyrotropic hormone in the urine and observations of the developmental response of the patient to thyroid and pituitary medication. It is hoped that the assays of the thyrotropic hormone may permit differentiation of primary hypothyroidism from hypothyroidism secondary to pituitary deficiency. The lack of proof of a specific pituitary growth hormone is pointed out.

DISCUSSION

DR. JOHN D. LYTTLE, New York: I should like to ask Dr. Wilkins if determinations of the blood cholesterol were of any help in regulating treatment.

DR. LAWSON WILKINS, Baltimore: My associates and I believe that there is a definite relationship of the level of the blood cholesterol to the activity of the thyroid gland. We have recently carried out studies on a number of dwarfs of the various types described, following the creatine output, the level of the blood cholesterol, the basal metabolic rate and the blood iodine curve. We hope to establish definite diagnostic criteria which will enable us to say which of these dwarfs have hypothyroidism and which have not, but so far we are not ready to report the results of these studies.

In regard to cholesterol, we have seen in many instances that discontinuance of thyroid therapy has caused the blood cholesterol to rise—in the dwarfs often

to a level of 300 or 400 mg. per hundred cubic centimeters. In cretins we have seen it rise to over 1,000 mg. With institution of thyroid therapy and gradual stepping up of the dose, in all the instances we have been able to follow so far, the blood cholesterol has dropped from levels between 300 and 400 mg. to levels between 100 and 125 mg. In regard to the clinical value of the determination of cholesterol in regulating dosage, it is probably only of secondary value. If one found the cholesterol high, I think one could be reasonably sure that not sufficient thyroid was being given. However, if the cholesterol is within normal limits, it is still possible that the dose is inadequate to accelerate the developmental rates to the desired degree.

In following the treatment, the important thing is to try to push thyroid to the point of mild hyperthyroidism and to follow the developmental curves, as I have shown, with the idea of bringing the level of development as close to normal as possible.

Relation of Endocrine Disturbances to Certain Heredodegenerative Symptoms. Dr. A. Graeme Mitchell and Dr. Josef Warkany (by invitation), Cincinnati.

One of the great problems in clinical endocrinology is the fact that with the endocrine defect is associated a multiplicity of symptoms. The difficulty lies in determining whether all of the accompanying symptoms are the result of, the cause of, or merely coincident with, the endocrine condition. There are discussed, first, diseases in which endocrinopathy may be the cause of certain conditions, such as myopathies and some bone diseases. Then are noted conditions in which a nonendocrine symptom may be considered as the cause of an endocrine dysfunction. In this connection different types of infantilism are mentioned. Finally, cases in which two endocrine conditions exist together are detailed, and the question of their causal connection is raised. The practical importance of these questions for the diagnosis and treatment of endocrine malfunctions in children is pointed out, and possible ways to investigate whether or not causal connections exist between heredodegenerative deviations and simultaneous endocrine abnormalities are suggested. Lantern slides illustrate certain parts of the discussion.

DISCUSSION

Dr. Julian D. Boyd, Iowa City: Dwarfism is not uncommon in diabetic children. Frequently it is assumed that the condition is dependent on some endocrine dysfunction. Careful study of such children makes it seem unlikely that their retardation is to be attributed to anything other than undernutrition. Reviewing records of the growth of 165 children with diabetes, I found that 10 had heights which were three deviations or more below the standards for their respective ages. With one exception, the retardation in growth had become established during long periods of inadequate dietary allowance or of uncontrolled diabetes. When adequate dietary allowance was enforced, resumption of growth was the rule, tending to restore the child to normal proportions. The one exception was observed in a true dwarf, small from birth, with a small sella turcica; the diabetes of this child seemed an additive, not a causative, factor.

Evidence demands that one assume that the child with diabetes has the capacity for normal growth. If this child does not grow at a normal rate, one should challenge the dietary therapy employed. Until the long-continued effects of adequate diet and good control of the diabetes have been established, there seems to be no excuse for administering pituitary or other growth-promoting products.

Food Requirements of Young Infants. Dr. Charles Hendee Smith, New York.

In many published papers and textbooks there is fairly general agreement as to the food requirements of infants as follows:

The total caloric needs are about 45 calories per pound (100 per kilogram) through the first year and rarely over 50 calories per pound. The protein require-

ments are satisfied by 1½ to 2 ounces of milk per pound (yielding 3.5 to 4.5 Gm. of protein per kilogram). It is necessary to add only from 1/12 to 1/10 ounce of sugar per pound (from 5.5 to 6.6 Gm. of sugar per kilogram) and rarely over 1 ounce a day. The fluid volume needed is from 2½ to 3 ounces per pound (from 165 to 200 cc. per kilogram) in the early months and not over 2 ounces per pound (130 cc. per kilogram) in the later months.

In recent years certain writers have insisted on a plan of giving concentrated feedings of high caloric value. Whole milk plus sugar up to from 8 to 10 per cent or evaporated milk mixtures of equal strength are dogmatically stated to be greatly superior to the feeding outlined in this paper. The addition of 2 or even 3 ounces of sugar is recommended, and an energy quotient as high as from 55 to 60 calories per pound (from 120 to 130 calories per kilogram). These concentrated mixtures contain 30 calories or more to 1 ounce.

Drug houses, evaporated milk companies and makers of certain sugars have seized the view of the advocates of this newer plan and have widely circularized the medical profession in attractively printed and convincing form. The general practitioner, obstetrician or inexperienced pediatrician may easily be misled by these presentations.

The proponents of this concentrated feeding say that the infant will not take more than he needs but will automatically cut down his own intake. Unfortunately, this is not always true. The baby with a good appetite will usually take all he can get, and overfeeding may easily result. In New York a good many babies are being definitely upset by too much food. They show this by failing appetite or by colic with crying, vomiting, loose stools, sometimes with fever. Others get too fat, and eczema is apt to develop.

But it is in the first weeks of life that the greatest damage is done. The infant gets very little breast milk in his first days. Toward the end of the week he may obtain from 25 to 30 calories per pound (from 55 to 66 per kilogram) and in the second week will gain on from 35 to 40 calories per pound (from 77 to 88 per kilogram). Not until the third week does the breast milk supply 45 calories per pound (100 per kilogram), yet the publications mentioned recommend mixtures which give from 55 to 60 calories per pound *from the day of birth.* In many small hospitals a "standard" formula for the new-born is of this strength.

Many obstetricians order formulas which are either too strong or too rapidly increased. Often the baby does well enough in the hospital, but when he is turned over to the pediatrician at the end of from two to four weeks he is beginning to show distress. This is the beginning of many difficult problems in feeding.

It seems logical to give a newly born infant who must be fed by bottle no more food than he would normally get from the breast. The prime objects should be to educate his digestion to artificial food, to avoid upsets at any cost and finally to establish a gain in weight. These objects are easily attained if one gives a fairly dilute mixture in the first week (from a fourth to a third milk), and gradually increases the milk in the second week to half strength. In these weeks ½ to ¾ ounces (from 15 to 25 Gm.) of sugar is enough.

Evidence is presented and will be published later, showing that breast-fed and bottle-fed infants gain on diets low in calories in the first weeks and also that growth is adequate throughout the year on between 45 and 50 calories per pound. Cases illustrating the bad results of early overfeeding are also cited.

DISCUSSION

DR. FRITZ B. TALBOT, Boston: Dr. Smith's paper is timely. The question comes to my mind what kind of babies are wanted. Are obese giants desired or not? I see babies develop into great stupid giants because they are overfed. I think Dr. Smith has presented this subject very well, but I disagree with him about one method of presentation. I have said it so many times before this society that I hope you will forgive me if I say it again, that I am not in sympathy with estimating the calories per kilogram. The reason is that this method of computing food needs and also figures for the basal metabolism often leads to unwarranted conclusions.

For example, if for some reason or other one has a fat baby with very little muscle and one gives it 45 calories per kilogram one may be giving the baby very much too much. If one has a very thin baby and gives him only 45 calories per kilogram one gives him too little food. In all of these babies the total caloric intake should be known to prevent mistakes.

There is one other thing I should like to repeat. I shall never forget a conversation I had with John Howland when I was first starting to learn how to feed babies. The older members of this society remember the hot arguments that took place on these floors in regard to infant feeding. John Howland came out of one of these meetings one day and said, "It does not make very much difference what you feed a baby as long as you know how to feed it." Dr. Smith was really emphasizing that, because Dr. Smith knows how to feed babies, but I am afraid some of the younger men who have not had as much experience have not run into the various pitfalls of feeding.

DR. HENRY DIETRICH, Los Angeles: I am in sympathy with Dr. Smith's presentation. He made an excellent point that one is apt to forget, namely, that under all circumstances one should avoid difficulty during the neonatal period. That is too frequently forgotten. If one upsets these young children during the first two, three or four weeks, what may appear to be a relatively small upset may develop into an emergency before one gets through.

All know that the infant fortunately has a very wide tolerance for food. If that were not true, many of the members of this society would not be here. Therefore, the mere fact that the child will take these highly concentrated mixtures certainly cannot be construed as meaning that they are good for the child. It is not a question of determining the optimum amount a child can take. The question is: What is the proper dilution or the proper concentration under which the child will make the most favorable progress, especially during the neonatal period?

DR. CHARLES HENDEE SMITH, New York: Since the discussion has been brief I should like to ask permission to include in the proceedings quotations from the writings of some of those present. I believe that the increasing tendency to overfeed new-born babies is killing babies, in New York at least.

Morse and Talbot (1915): "If the baby has to be given artificial food, it is important not to give too strong a mixture at first. If the baby is upset by too strong a food, it is a difficult matter to correct the disurbance."

Hess: "During the first weeks of life lesser quantities of food must be given than is recommended for later periods."

Holt and McIntosh: "The first days' requirements are low, sixty calories per kilo. In the second and third week they rise rapidly to 100 calories per kilo."

Brennemann: "The first two weeks stand out precariously, in striking contrast to later infancy. A good start is of vital importance and largely determines the later course."

Marriott: "The first week or two the metabolism is relatively low. Only in the second or third week does it rise."

Schick: "Concentrated feeding should not be used for normal infants but only for special indications."

It may serve a good purpose to have these quotations printed in the proceedings of this society as a reminder that it is not good to give a baby too much food in the early days of life at least.

I am sure Dr. Talbot is mistaken in his reading of the weight charts shown. None of these infants had too much food, never over 50 calories per pound, usually 45 or less.

(To Be Concluded)

Book Reviews

Physiologie und Pathologie des Wasserhaushaltes. By Dr. Ladislaus v. Kostyál. Price, 11 marks. Pp. 160, with 10 figures and 1 table. Berlin: S. Karger, 1936.

In the short space of 160 pages the author of this monograph presents a fairly comprehensive review of present day conceptions regarding the water economy of the body under both physiologic and pathologic conditions. As indicated by the bibliography of 1,192 papers, the treatise is largely a review of known facts and theories, although some original data are presented.

After a short discourse on the peculiar physical and chemical properties of water, which make it especially fit for its varied rôles in the living body, the author describes the various forms in which water occurs in the body. These he designates as follows : (1) that recognized as water of solution, (2) that loosely bound with colloids by a reversible reaction, (3) that which is chemically bound and (4) that which occurs in certain pathologic states, spoken of as free extracellular water. These forms are not clearly differentiated by the author. The partition of water between the various organs is given. The major part of the monograph is devoted to discussion of the physiologic aspects of water metabolism. The factors governing translocation, or exchange of water between different compartments, are described in detail. The forces responsible for absorption of water from the gastro-intestinal tract into the blood stream, across the capillary wall and across the cell membrane are pointed out. The author concluded from the work of Heidenhain, Asher and certain other investigators that the passage of water from the blood stream to the tissues and vice versa cannot be accounted for on a simple physical basis but must be explained on the basis of special activity of the endothelial cells. The special rôles of salts and other crystalloids and also of various colloids in relationship to osmotic pressure effects are reviewed. The physical nature of colloids and the manner in which water is held by hydrophilic colloids are described. The special importance of the plasma proteins and of the blood and tissue lipids in binding water is stressed.

The special rôles of the various organs and parts of the body in regulating the movements of water are discussed individually. This discussion includes consideration of the specific functions of the various endocrine glands, as well as of the function of the central and the vegetative nervous system and of the heart, lungs, kidneys, liver and spleen. While the rôle of the circulatory system and lungs is chiefly mechanical, the endocrine glands indirectly exert physicochemical effects involving such factors as the permeability of cell membranes, the specific metabolism of cells and the exchange of minerals. The nervous system probably exerts a direct influence on the permeability of cell membranes as well as an inhibitory or stimulatory effect on organ systems such as the endocrine glands and the cardiovascular system.

The effects of diet, of previous water intake and of posture on the storage or excretion of water are considered. The parallelism between the total energy and the water exchange is noted. Several authors, who are quoted, agree that the infant's intestinal wall is more permeable to water than that of the adult and that this permeability is increased as a result of nutritional disturbances. Adequate proof for such assertions is not given.

In the less comprehensive section, that on the water economy under pathologic conditions, the author considers disturbances in water metabolism under four main groups, namely, (1) conditions characterized by pathologic retention of water, (2) conditions the reverse of these, in which the body suffers from a deficiency of water, (3) febrile states in which the water exchange is greatly modified as a result of the fever itself and (4) conditions in which the water balance is abnormal as a result of dysfunction of the endocrine glands and

nervous system. Under the first heading are discussed the various forms of edema, with special reference to their mechanical and physiochemical mechanisms. Circulatory, renal, postural and protein starvation factors are emphasized. The significance and limits of extrarenal loss of water and crystalloids in the presence of edema are discussed. Under the second heading the author considers the mechanism of water loss in connection with diarrhea, vomiting and excessive sweating and points out the abnormal responses of the various structures of the body with regard to the loss and the retention of salt as a result of the primary disturbance. It is pointed out that water and salts are often retained to an abnormal degree in the early phases of a febrile illness and that normal balances are restored as convalescence approaches. Disturbances in the water and salt balances of the organism as secondary phenomena in connection with disorders of the pituitary, thyroid, adrenal and other endocrine glands are then considered briefly. Finally, the applications of the fundamental principles brought out are summarized in connection with a discussion of nutritional and allied disorders in infants and young children.

La diphtèrie. By B. Carrière. Price, 35 francs. Pp. 214, with 49 illustrations. Paris Masson & Cie, 1936.

Carrière is professor of medicine at the University of Lille. He writes clearly and concisely and reveals himself a careful observer and investigator. He has had the opportunity of studying large numbers of patients with diphtheria and is well fitted to describe not only the usual clinical picture of the disease but also the more rare manifestations. The effect of associated infections on the course of diphtheria are considered and also the manifestations of the diphtheria which develops in the course of other infections, such as scarlet fever and measles. Considerable attention is paid to the disturbances of the vascular system associated with diphtheria, and there is a brief discussion of the various forms of paralysis which may develop.

In discussing serum therapy, the author expresses the opinion that massive doses may be harmful and states definitely that 500 units of antitoxin per kilogram of body weight is sufficient. Theoretically, a single injection should be all that is necessary, but if the membrane spreads and the general condition does not improve, equal or slightly larger doses may be given during succeeding days.

The indications for and the technic of intubation and tracheotomy are given in detail. The last two chapters describe various therapeutic measures which may add to the comfort of the patient and reenforce the beneficial effects of the antitoxin.

Clinical Psychology. By C. M. Louttit, director of the Indiana University Psychological Clinics. Price, $3.50. Pp. 695, with 20 illustrations and 85 tables. New York: Harper & Brothers, 1936.

This book covers the whole field of clinical psychology. In the introduction the author defends the nonmedical psychologist as capable of treating behavior disorders. He uses the term psycho-clinician to designate such a person.

Diagnostic methods—the taking of the history and the physical measurements and the performance of psychometric tests—are carefully described. The problems of mental retardation and retardation in school are considered, as well as special disabilities in school. A chapter is devoted to superior children.

Nearly one half of the book is devoted to behavior problems. This includes a consideration of the factors and attitudes which contribute to these problems. The common symptoms of misbehavior are described. The explanatory matter consists mostly of general and objective descriptions of conditions, with data on their frequency. The exact emotional problems underlying the condition are usually not explained.

The recommended treatment of enuresis consists in restriction of fluids and periodic awakening during the night. Masturbation in young children "probably

has no sexual significance." Juvenile delinquency is discussed in general, but the specific problems of the adolescent are omitted. The neuroses, psychoneuroses and organic disorders are briefly described.

"This book is intended as a textbook for students of clinical psychology." For undergraduate college students it should be of great value.

Feeding, Diet and the General Care of Children. By Dr. Albert J. Bell. Price, $2. Pp. 316, with 7 illustrations. New York: G. P. Putnam's Sons, 1937.

This book meets a demand which many mothers and nurses without special pediatric training may feel. Definite instructions are given as to breast feeding, the preparation of artificial foods, the clothing and bathing of the child and such procedures as lavage, gavage and the giving of enemas. There are also chapters on the recognition and general care of acute infections and chronic conditions, e. g., tuberculosis. It may appear to some persons that too much information is given, but it surely seems right that a mother should know, for example, of the possibility of pyloric stenosis so that she will not take her baby from the breast in a vain effort to find a food which will better agree with it and that she should have some knowledge of the symptoms of intussusception so that she may not delay in receiving medical advice. The chapter "Some Aspects of Child Behavior" gives an understanding of the psychologic development of the child which will prevent much despair on the part of the mother and consequent ill directed training.

It is remarkable that so much material can be covered in so small a book. Of necessity, the presentation is often didactic. Questions at the end of each chapter accentuate this character. However, the information is accurate and the attitude developed and the emergency procedures described may prove of inestimable value. The book is well indexed, which is often not the case in similar works.

Der jugendliche Schwachsinn. By Wilhelm Weygandt. Price, paper, 28 marks; bound, 30 marks. Pp. 428, with 159 illustrations. Stuttgart: Ferdinand Enke, 1936.

The literature on mental deficiency is so vast that one finds himself in a maze when he tries to construct a comprehensive picture of all the many phases of this unfortunate condition. There are, to be sure, other monographs on mental deficiency, but in the eyes of the reviewer none as all embracing as this. For one finds that few details were omitted from this volume. The contents are divided into two main parts.

In part I, the first chapter deals with historical phases of mental deficiency. There are also chapters on the normal physical and mental development of the child, with a discussion on precocity and genius. The chapters on causes of mental deficiency, psychic symptoms, anatomic stigmas, pathologic changes observed at postmortem examination, diagnosis and prognosis, prevention and educational therapy are admirable.

In part II are discussed the clinical phases, viz., the endogenous and exogenous disturbances manifested in mental deficiency, such as anencephaly, microcephaly, hydrocephaly, amaurotic familial idiocy, Friedreich's ataxia, muscular dystrophy, epilepsy, mongolism, meningitis, endocrine disturbances, vascular disease and conditions due to trauma.

Generally, the book is well written and the German literature well covered. The illustrations and charts are instructive.

Vermeidbare Kinderkrankheiten. By Dr. Walter Birk. Price, paper, 12 marks; bound, 13.60 marks. Pp. 232, with 21 illustrations. Stuttgart: Ferdinand Enke, 1936.

The content of the book is much more inclusive than would be anticipated from the title. Much of the material (such as a lengthy discussion of the low incidence

of breast feeding, nutrition and dietary fads) seems irrelevant. The importance of breast feeding is duly emphasized. More space is devoted to accounts of diseases and abnormal conditions than to their prevention. An excellent description of tuberculosis in children is presented. The advice relating to child guidance and the management of the problems of adolescence is also good.

Regarding the prevention of disease, several phases are brought out. The importance of individual and family responsibility and cooperation with the state are stressed. Well recognized general principles of preventive medicine, such as avoidance of exposure to disease (especially at certain age periods) and instruction of the laity are discussed. In connection with hereditary diseases, the merits and shortcomings of genetics are pointed out.

Some of the author's views concerning the prevention of specific disease is conservative from the standpoint of American pediatrics. Active immunization against scarlet fever and pertussis is not mentioned, and that against diphtheria is considered but rejected.

The book can be recommended as interesting reading matter but not as a scientific reference book on the subject.

Slovensko Bez Dorastu? By Prof. Dr. A. J. Chura. Pp. 464, with no illustrations. Bratislava: Tlacila Slov. Grafia, 1936.

This is a statistical summary of the condition of children in Slovakia, the eastern province of Czechoslovakia. The text is in the Slovakian language, with the résumés in German and French.

The author discusses in detail the infant mortality and the birth rate of this province as compared with those of Bohemia and Moravia. The birth rate has fallen continuously, but is higher in Slovakia than in the other provinces. The death rate of children in Slovakia, 145.3 per thousand infants alive at birth, is high, much higher than that of Bohemia. In studying the situation, the author found that the majority of deaths were of new-born infants, and he ascribed them to lack of skill in obstetrics and to diarrheal diseases, which have not decreased in Slovakia in many years. This, he thinks, is due to the fact that sufficient effort has not been made to help the families and to promote public health. Other factors in this high rate are tuberculosis and diseases of the respiratory tract.

Synopsis of Pediatrics. By John Zahorsky, M.D., Professor of Pediatrics and Director of the Department of Pediatrics, St. Louis University School of Medicine, and T. S. Zahorsky, M.D., Assistant in Pediatrics, St. Louis University School of Medicine. Second edition. Cloth. Price, $4. Pp. 367, with 89 illustrations. St. Louis: C. V. Mosby Company, 1937.

The present edition of this little book is similar to the first (1934) except for a moderate increase in the list of subjects considered and a slight amount of revision of the original text. It is a pocket-sized volume, well printed and securely bound, and contains eighty illustrations in black and white and nine colored plates.

It is primarily intended for the use of medical students and offers them a brief yet fairly comprehensive presentation of the most important facts concerning the health and the diseases of infants and children. In addition, it should be of service to teachers because of the arrangement of the material into sixty-one chapters, each of which is suitable for an hour's work in the classroom, and of value to busy general practitioners because of the careful enumeration of the essential points on symptomatology, diagnosis and treatment of disease. The book, however, is in no way intended to replace larger textbooks with fuller discussions and references to pediatric literature.

The general field of pediatric subjects is well covered. Subjects which are purely pediatric receive more extended consideration than those which are shared by other specialties, such as neurology, dermatology and orthopedics. The communicable diseases are fully discussed. The treatment of the various topics

naturally is to a large extent didactic, but owing to the authors' happy faculty for terseness of expression an amazing amount of information is packed into each paragraph.

In spite of the evident effort to condense the material as much as possible, the book is extremely readable, and its interest is enhanced by the interspersion of concise and pointed statements of the personal opinions of the authors.

Recent Advances in Orthopædic Surgery. By B. H. Burns, B. A., B.Ch., F.R.C.S., and V. H. Ellis, M.A., B.Ch., F.R.C.S. Cloth. Price, $5. Pp. 296, with 108 illustrations. Philadelphia: P. Blakiston's Son & Co., 1937.

The authors have reviewed, in a small volume, a fairly representative cross-section of the more recent contribution to medical and scientific literature in the field of orthopedic surgery. The chapters dealing with the chemistry of bone and the theories of bone growth are exceptionally noteworthy.

The statement is made in the preface that as far as possible the authors have dealt with the application of principles rather than with the details of technic. American surgeons may not agree that they are justified in the emphasis they place on some of the principles of treatment, such as those described for tuberculosis of the joints. Little that is new about osseous tuberculosis is to be found in the three chapters, covering thirty pages, and although various types of surgical procedures, most of which have been used successfully in this country for at least twenty-five years, are mentioned, almost equal space is taken by descriptions of methods of traction and countertraction which are certainly not new and have for the most part been abandoned as inadequate or ill advised.

Although a chapter devoted to adolescent coxa vara includes a discussion of Legg-Calvé-Perthes' disease (the authors speak of the condition as Perthes' disease), no mention is made of the important contribution of Danforth in demonstrating the successful treatment of this entity and the prevention of deformity.

In spite of some deficiencies from the standpoint of selections from the literature or critical consideration of the material presented, much in the volume will be helpful to the orthopedic surgeon. It may also be used as a brief reference work by the general surgeon or the physician in general practice who has not had the opportunity of reading most of the current journals dealing with orthopedic surgery during the last few years.

Neurología Infantil. By A. Gareiso and F. Escardo. Paper. Price not given. Pp. 134. Buenos Aires: El Ateneo Libreria Cientifüca Y Lit, 1936.

This book is based on the authors' studies of 560 cases of epilepsy, 439 cases of mania, 181 cases of encephalopathy, 86 cases of Little's disease and 81 cases of hemiplegia. For epilepsy, syphilis was the cause in 39 per cent of the cases, obstetric trauma in 15 per cent, prematurity in 1.5 per cent and alcoholism in 2.5 per cent. The cause was unknown in 40 per cent. The proportion was practically the same for mania. For encephalopathy, syphilis was the cause in 42 per cent of the cases, trauma in 29 per cent and prematurity in 13 per cent. The cause was unknown in 28 per cent. For hemiplegia, syphilis was the cause in 40 per cent of the cases, trauma in 33 per cent, alcoholism in 2.5 per cent and prematurity in 5 per cent. For Little's disease, syphilis was the cause in only 23 per cent of the cases, while obstetric trauma was the cause in 50 per cent and prematurity in 40 per cent.

These cases provide excellent material for study, and the authors have developed it well. There are chapters on pathology and prophylaxis. Although in Spanish, this book should be of great value to any one interested in this phase of pediatrics.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Professor G. Scheltema, 6a Herreplein, Groningen, Netherlands.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Prof. Luigi Spolverini, Policlinico Lungo Tevere Melini, 24, Rome, Italy.
Secretary: Professor Vitetti, Rome, Italy.
Place: Rome, Italy. Time: Sept. 27-30, 1937.
American Committee:
Chairman: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary: Dr. L. Emmett Holt Jr., Johns Hopkins Hosp., Baltimore.
Canadian Committee:
Chairman: Dr. H. B. Cushing, 1390 Sherbrooke St., W., Montreal.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St., W., Montreal.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.
Place: Rome, Italy. Time: Oct. 4-8, 1937.

FOREIGN

British Paediatric Association

President: Dr. Leonard Findlay, 14 Wimpole St., London, W. 1.
Secretary: Dr. Arthur G. Maitland Jones, 31A Weymouth St., London, W. 1.

Paediatrický Spolok na Slovensku

President: Dr. A. J. Chura, Lazaretská 6, Bratislava, Czechoslovakia.
Secretary: Dr. S. Limbacher, Dlhá 2, Bratislava, Czechoslovakia.
Place: Pediatric Clinic, University, Bratislava. Time: Six times a year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Sir Lancelot Barrington-Ward, 85 Harley St., London, W. 1.
Secretary: Dr. K. H. Tallerman, 122 Harley St., London, W. 1.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:30 p. m.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialikstr., Tel Aviv, Palestine.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv, Palestine.

Sociedad Cubana de Pediatría

President: Prof. Félix Hurtado Galtés, Carlos III, 7, Havana.
Secretary: Dr. Carlos Hernández Miyares, San Lazaro 370, Havana.
Place: Cátedra de Clínica Infantil, Hospital Mercedes, Havana. Time: Third Thursday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. Clifford D. Sweet, 2940 Summit St., Oakland, Calif.
Secretary: Dr. Albert D. Kaiser, 16 N. Goodman St., Rochester, N. Y.
Place: San Francisco. Time: June 13-17, 1938.

American Academy of Pediatrics

President: Dr. Philip Van Ingen, 125 E. 71st St., New York.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Region I. Place: Boston, Mass. Time: Nov. 12-13, 1937.
Region II. Place: New Orleans, La. Time: Nov. 30-Dec. 1-3, 1937.
Region III. Place: Lake Shore Athletic Club, Chicago. Time: Oct. 14-16, 1937.
Region IV. Place: Los Angeles, Calif. Time: October 1937.

American Hospital Association, Children's Hospital Section

Chairman: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Secretary: Miss Edith F. Bateman, Shriners' Hospital for Crippled Children, St. Louis, Mo.

American Pediatric Society

President: Dr. Kenneth D. Blackfan, 300 Longwood Ave., Boston.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid St., St. Louis.
Place: The Sagamore, Bolton Landing, New York. Time: June 9-11, 1938.

Canadian Society for the Study of Diseases of Children

President: Dr. Alton Goldbloom, 1543 Crescent St., Montreal, Quebec.
Secretary-Treasurer: Dr. Pearl Summerfeldt, Hospital for Sick Children, Toronto, Ont.

Society for Pediatric Research

President: Dr. Francis Scott Smyth, University of California Medical School, San Francisco.
Secretary: Dr. A. A. Weech, 3975 Broadway, New York.

SECTIONAL

North Pacific Pediatric Society

President: Dr. D. M. Dayton, Medical Arts Bldg., Tacoma, Wash.
Secretary: Dr. H. T. Clay, Medical Arts Bldg., Tacoma, Wash.
Place: Portland, Ore. Time: June 1937.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. Gilbert Levy, 20 S. Dunlap St., Memphis, Tenn.
Secretary: Dr. Hughes Kennedy Jr., Highland Plaza Apts., Birmingham, Ala.

STATE

Alabama Pediatric Society

President: Dr. Wallace A. Clyde, Employees' Hospital, Fairfield.
Secretary-Treasurer: Dr. M. G. Neely, Employees' Hospital, Fairfield.

Arkansas State Pediatric Association

Chairman: Dr. A. C. Kirby, 701 Main St., Little Rock.
Secretary: Dr. Madeline M. Melson, 909 Main St., Little Rock.

California State Medical Society, Section on Pediatrics

Chairman: Dr. E. Earl Moody, 3780 Wilshire Blvd., Los Angeles.
Secretary: Dr. Hartzel H. Ray, 23 Second Ave., San Mateo.

Florida State Pediatric Society

President: Dr. Douglas D. Martin, 442 W. Lafayette St., Tampa.
Secretary: Dr. Warren W. Quillian, 227 Avenue Aragon, Miami.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St., N. E., Atlanta.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Howard W. Brayton, 179 Allyn St., Hartford.
Secretary: Dr. Joseph L. Hetzel, 103 N. Main St., Waterbury.

Indiana State Pediatric Society

President: Dr. Ernest R. Carlo, 2902 Fairfield Ave., Fort Wayne.
Secretary-Treasurer: Dr. Mathew Winters, 23 E. Ohio St., Indianapolis.
Time: Two meetings a year.

Louisiana State Pediatric Society

President: Dr. Clarence H. Webb, 624 Travis St., Shreveport.
Secretary-Treasurer: Dr. Ruth Aleman, 1407 S. Carrollton Ave., New Orleans.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. George M. Retan, 459 James St., Syracuse.
Secretary: Dr. John D. Craig, 33 E. 61st St., New York.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Secretary: Dr. C. R. Dengler, 131 W. Michigan Ave., Jackson.

Mississippi State Pediatric Society

President: Dr. Guy C. Jarratt, 920 Cawford St., Vicksburg.
Secretary-Treasurer: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Place: To be designated by Society. Time: Yearly.

Nebraska Pediatric Society

President: Dr. J. A. Henske, 1614 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. H. Murphy, 915 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. John LaBruce Ward, Arcade Bldg., Asheville.
Secretary: Dr. Lewis Elias, 36 Grove St., Asheville.

Oklahoma State Pediatric Society

President: Dr. B. H. Nicholson, 301 N. W. Twelfth St., Oklahoma City.
Secretary: Dr. Hugh C. Graham, 1501 S. Baltimore Ave., Tulsa.

Pennsylvania Medical Society, Pediatric Section

Chairman: Dr. Theodore O. Elterich, 3700 Fifth Ave., Pittsburgh.
Secretary: Dr. John M. Higgins, Sayre.

South Carolina Pediatric Society

President: Dr. Leland B. Salters, 317 S. Warley St., Florence.
Secretary-Treasurer: Dr. D. Lesesne Smith Jr., 187 Oakland Ave., Spartanburg.

TENNESSEE STATE PEDIATRIC SOCIETY

President: Dr. Joe T. Smith, 702 Medical Arts Bldg., Knoxville.
Secretary: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis.

TEXAS PEDIATRIC SOCIETY

President: Dr. John G. Young, 3930 McKinney Ave., Dallas.
Secretary-Treasurer: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Place: Dallas. Time: Fall.

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Basil B. Jones, 1028 W. Franklin St., Richmond.
Secretary: Dr. W. A. McGee, 616 W. Grace St., Richmond.

WEST VIRGINIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

President: Dr. John T. Thornton, 58 16th St., Wheeling.
Secretary: Dr. Claude L. Holland, 400 Locust Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. J. D. Pilcher, City Hosital, Cleveland.
Secretary: Dr. William M. Champion, 2323 Prospect Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. T. B. Verner, 170 St. George St., Toronto.
Secretary: Dr. D. V. Hutton, 170 St. George St., Toronto.

BRONX PEDIATRIC SOCIETY

President: Dr. Daniel J. Doan, 420 E. 159th St., New York.
Secretary: Dr. Samuel T. Levethan, 254 E. 184th St., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

BROOKLYN ACADEMY OF PEDIATRICS

President: Dr. Walter D. Ludlum, 1421 Cortelyou Rd., Brooklyn.
Secretary: Dr. John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Fourth Wednesday of each month, except May, June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vasder Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Thursday of April and September.

CHICAGO PEDIATRIC SOCIETY

President: Dr. C. A. Aldrich, 723 Elm St., Winnetka, Ill.
Secretary: Dr. George F. Munns, 723 Elm St., Winnetka, Ill.
Place: Children's Memorial Hospital, 710 Fullerton Ave., Chicago. Time: Third Tuesday of each month, October to May, inclusive.

DALLAS PEDIATRIC SOCIETY

President: Dr. John Young, 3930 McKinney Ave., Dallas, Texas.
Secretary-Treasurer: Dr. John E. Ashby, 3622 Fairmont Ave., Dallas, Texas.
Place: Bradford Baby Hospital. Time: 1 p. m., second and fourth Saturdays of each month.

Detroit Pediatric Society

President: Dr. Edward A. Wishropp, 10 Peterboro St., Detroit.
Secretary: Dr. W. J. Scott, 5140 Second Ave., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month from October to June, inclusive.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. T. F. Davenport, 104 Ponce de Leon Ave., Atlanta.
Secretary: Dr. James P. Hanner, 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Houston Pediatric Society

President: Dr. Walter F. Qualtrough, 920 Westheimer Ave., Houston, Texas.
Secretary-Treasurer: Dr. Allan Bloxsom, 4402 Travis St., Houston, Texas.
Place: Houston Club. Time: Third Monday of each month.

Intermountain Pediatric Society

President: Dr. Eugene Smith, 385 Twenty-Fourth St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City, Utah.
Place: Salt Lake City, General Hospital. Time: First Thursday of each month, 8 p. m.

Kansas City (Missouri) Pediatric Society

President: Dr. W. R. Moore, 620 Francis St., St. Joseph.
Secretary: Dr. F. S. Hogue, 1103 Grand Ave., Kansas City.
Place: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Edwin F. Patton, 3875 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Henry F. Gallagher, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd.
Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Harry R. Litchfield, 163 Eastern Parkway, Brooklyn.
Secretary: Dr. Jacob Rosenblum, 550 Ocean Ave., Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: Third Wednesday of each month, October to May.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. W. C. A. Steffen, 42 Union St., Flushing, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144 38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Memphis Pediatric Society

President: Dr. Clyde V. Croswell, 1052 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. David W. Goltman, 1073 Madison Ave., Memphis, Tenn
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. George F. Kelly, 425 E. Wisconsin St., Milwaukee.
Secretary: Dr. Helen Jane Zilmer, 781 N. Jefferson St., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

New England Pediatric Society

President: Dr. Henry E. Utter, 122 Waterman St., Providence, R. I.
Secretary-Treasurer: Dr. Henry E. Gallup, 1101 Beacon St., Brookline, Mass.
Place: Boston Medical Library. Time: Four meeting a year, occurring from September to May.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Alexander T. Martin, 107 E. 85th St., New York.
Secretary: Dr. Philip M. Stimson, 25 Claremont Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northwestern Pediatric Society

President: Dr. A. V. Stoesser, University Hospital, Minneapolis.
Secretary: Dr. Roger L. J. Kennedy, Mayo Clinic, Rochester, Minn.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Oklahoma City Pediatric Society

President: Dr. A. L. Salomon, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 1200 N. Walker St., Oklahoma City.
Time: Alternate months.

Philadelphia Pediatric Society

President: Dr. Emily Bacon, 2104 Spruce St., Philadelphia.
Secretary: Dr. James E. Bowman, 4091 Comly St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. James LeRoy Foster, Highland Bldg., Pittsburgh.
Secretary-Treasurer: Dr. E. R. McClusky, 3200 Fifth Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. Lee E. Sutton Jr., 2601 Grove Ave., Richmond, Va.
Secretary-Treasurer: Dr. Louise Fry Galvin, 2416 Stuart Ave., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: First Thursday of each month (except July, August and September).

Rochester Pediatric Society

President: Dr. F. W. Bush, 29 N. Goodman St., Rochester, N. Y.
Secretary: Dr. David Wolin, 16 N. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: Second Friday of each month.

Rocky Mountain Pediatric Society

President: Dr. James E. Russell Jr., 820 Metropolitan Bldg., Denver.
Secretary: Dr. F. Craig Johnson, 1850 Gilpin St., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 2:30 p. m.

St. Louis Pediatric Society

President: Dr. John Zahorsky, 536 N. Taylor Ave., St. Louis.
Secretary-Treasurer: Dr. Max Deutch, 634 N. Grand Blvd., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

Seattle Pediatric Society

President: Dr. Frank H. Douglass, Medical Dental Bldg., Seattle.
Secretary: Dr. C. Lu Verne Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Howard R. Cooder, 3875 Wilshire Blvd., Los Angeles.
Secretary: Dr. Philip E. Rothman, 3875 Wilshire Blvd., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. M. Cooperstock, Northern Michigan Children's Clinic, Marquette.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. Louis V. Waldron, 149 N. Broadway, Yonkers, N. Y.
Secretary-Treasurer: Dr. Fairfax Hall, 421 Huguenot St., New Rochelle, N. Y.
Place: Grasslands Hospital. Time: Third Thursday, October, December, February and April.